Minnesota M

Contents fo

SCIENTIFIC ARTICLES

ORIGINAL CONTRIBUTIONS

CASE PRESENTATION

CONTINUATION STUDY

CLINICAL-PATHOLOGICAL CONFERENCE

Owner and Publisher

MINNESOTA STATE MEDICAL ASSOCIATION
496 Lowry Medical Arts Building
St. Paul 2, Minnesota

Publishing and Finance Committee

E. M. Hammes, M.D.
Chairman, Saint Paul

F. M. Owens, Jr., M.D.
Saint Paul

T. A. Peppard, M.D.
Minneapolis

Henry Ulrich, M.D.
Minneapolis

Business Manager

R. R. Rosell
496 Lowry Medical Arts Building
St. Paul 2, Minnesota

Official Journal of

Duluth Surgical Society
Minneapolis Academy of Medicine
Minneapolis Obstetrical and Gynecological Society
Minneapolis Surgical Society

Minnesota Academy of Medicine
Minnesota Academy of Occupational Medicine and Surgery
Minnesota Obstetric and Gynecologic Society
Minnesota Society of Clinical Pathologists

Minnesota Society of Internal Medicine
Minnesota Society of Neurology and Psychiatry
Minnesota State Medical Association
Minnesota Surgical Society

Minnesota Trudeau Society
Northern Minnesota Medical Association
Saint Paul Surgical Society
Southern Minnesota Medical Association

Minnesota M

Contents fo

SCIENTIFIC ARTICLES

ORIGINAL CONTRIBUTIONS

CASE PRESENTATION

CONTINUATION STUDY

CLINICAL-PATHOLOGICAL CONFERENCE

Owner and Publisher

MINNESOTA STATE MEDICAL ASSOCIATION
496 Lowry Medical Arts Building
St. Paul 2, Minnesota

Publishing and Finance Committee

E. M. Hammes, M.D.
Chairman, Saint Paul

F. M. Owens, Jr., M.D.
Saint Paul

T. A. Peppard, M.D.
Minneapolis

Henry Ulrich, M.D.
Minneapolis

Business Manager

R. R. Rosell
496 Lowry Medical Arts Building
St. Paul 2, Minnesota

Official Journal of

Duluth Surgical Society
Minneapolis Academy of Medicine
Minneapolis Obstetrical and Gynecological Society
Minneapolis Surgical Society

Minnesota Academy of Medicine
Minnesota Academy of Occupational Medicine and Surgery
Minnesota Obstetric and Gynecologic Society
Minnesota Society of Clinical Pathologists

Minnesota Society of Internal Medicine
Minnesota Society of Neurology and Psychiatry
Minnesota State Medical Association
Minnesota Surgical Society

Minnesota Trudeau Society
Northern Minnesota Medical Association
Saint Paul Surgical Society
Southern Minnesota Medical Association

ta Medicine

for February, 1958

Volume 41
Number 2

MISCELLANEOUS ARTICLES

ENERAL INFORMATION

thors

Manuscripts should be addressed to Arthur H. Wells, M.D., Editor, 915 East First Street, luth 5, Minnesota. Telephone RA 7-6636 or JA 5-2319.

aders and Reviewers

The right is reserved to reject material submitted for reading or advertising columns. e views expressed in this journal do not necessarily represent those of the Minnesota te Medical Association or any of its constituents.

vertisers and Subscribers

Classified advertising—15 cents a word; minimum charge $3.00; key number, 25c ditional. Remittance should accompany order. Display advertising rates on request.

Annual Subscription—$5.00. Single Copies—$0.50. Foreign and Canadian—$5.50.

Communications concerning advertising and subscriptions should be addressed to NNESOTA MEDICINE, 2642 University Avenue, Saint Paul 14, Minnesota. Telephone Mid- y 6-2641.

pyright and Post Office Entry

Contents of MINNESOTA MEDICINE ©1957 by Minnesota State Medical Association.

Entered at the Post Office in Saint Paul as second class matter. Accepted for mailing at special rate of postage provided for in Section 1103, Act of October 3, 1917, horized July 13, 1918.

Board of Editors

ARTHUR H. WELLS, M.D.
Editor-in-Chief, Duluth

STUART W. ARHELGER, M.D.
Minneapolis

JOHN F. BRIGGS, M.D.
Saint Paul

S. FRANCIS CEPLECHA, M.D.
Redwood Falls

TAGUE C. CHISHOLM, M.D.
Minneapolis

N. L. GAULT, JR., M.D.
Minneapolis

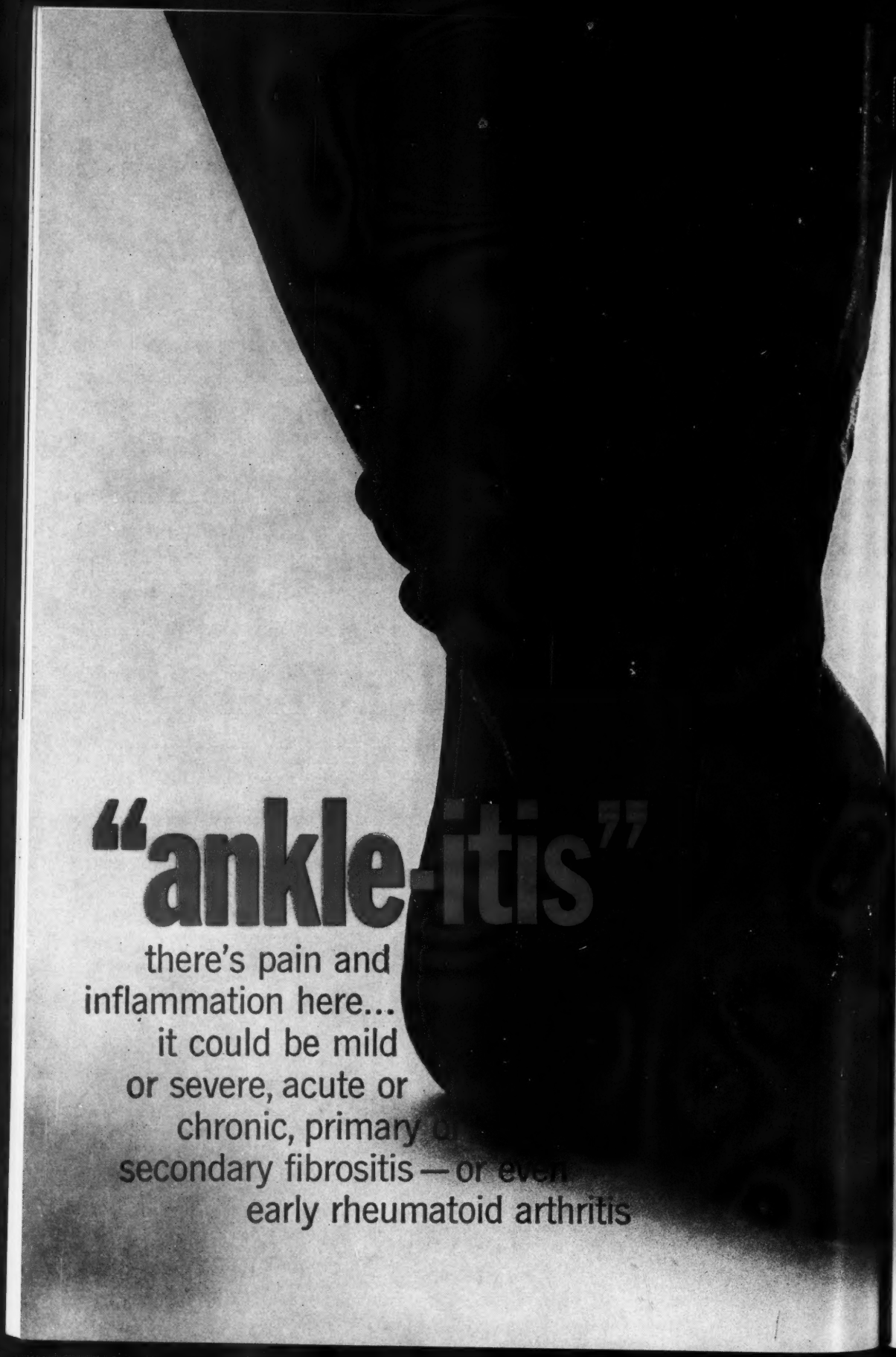
R. DREW MILLER, M.D.
Rochester

HENRY G. MOEHRING, M.D.
Duluth

GLENN J. MOURITSEN, M.D.
Fergus Falls

OLIVE V. SEIBERT, B.A.
Saint Paul

GEORGE STILWELL, M.D.
Rochester


"ankle-itis"
there's pain and
inflammation here...
it could be mild
or severe, acute or
chronic, primary
secondary fibrositis—or
early rheumatoid arthritis

Original Contributions

Acute Diarrhea of Infants and Small Children

A Review

THOMAS E. REICHELDERFER, M.D., M.P.H.

Bethesda, Maryland

DIARRHEA is not in itself a disease but rather a symptom of illness. It is characterized by frequent evacuation of the bowels with unformed stools, accompanied by large amounts of water, body electrolytes, and undigested food. It is well for this reason to discuss diarrhea as a single entity even though the etiology may vary from any of several bacterial agents, viruses, parasites, foods and emotional crises.

The treatment of diarrhea is directed primarily at the correction of the physiological disturbances produced by loss of water and ions, and secondly against the causative agent. Infants, because of the larger proportion of water making up the body mass, are more prone to suffer from severe dehydration and electrolyte loss than adults or older children. Three hundred to 500 cc. of water may be lost in the stools. Recent investigations have uncovered agents that are responsible for epidemic diarrhea of the newborn and parenteral diarrhea.[1]

The determination as to whether or not a child has diarrhea is usually a simple one. The parents report the use of an increased number of diapers soiled with a loose stool, accompanied by a large water ring. The child often asks to be placed on the pot but will frequently have an evacuation before the parent can undress him. The character of the stool in small infants is important. It is usually green, contains mucus and in the severe cases may be streaked with blood or be grossly bloody. The frequency is increased over the normal pattern for bowel movements. The transitional stool of the newborn with its green color often confuses young mothers sent home too early from the hospital. The lack of frequency and mucus and the change to yellow stools in a short time are often enough to reassure the mother and exclude diarrhea.

Since Colonial times, the summer with its high incidence of diarrhea (cholera infantum) has always been a particularly hazardous period for infants and small children. The author's grandmother, in the years following the Civil War, gave birth to thirteen children. Only six reached their majority. The remaining succumbed shortly after birth or during the first year of life to "summer complaint" or infantile diarrhea. Conditions changed little until about 1900. Diarrhea was at that time among the leading causes of death in the first year of life. The rates for deaths due to diarrhea and enteritis have fallen with the passage of time in those states which require registration. In 1900 the rate was 142.7 deaths per 100,000 population; 1920, 53.7; 1930, 2€; 1940, 10.3 and 1950, 4.9.[2]

The production of pasteurized and evaporated milks, refrigeration, pure water supplies and improved methods of sewage disposal have accounted in part for this reduction in infant mortality. The introduction of chemotherapeutic and antibiotic drugs and widespread use of parenteral fluids have brought about a further reduction in mortality due to diarrheal disease.

The known agents that cause gastroenteritis have changed little with the passage of time. They will always remain as a threat to our population. Some have become so rare that physicians rarely see a case caused by them. New causative agents are being discovered. The initial treatment in each is directed at the correction of physiological disturbances produced by the diarrhea. The following is a short description of some of the diarrhea-producing organisms, substances and states accompanied by a brief commentary.

Presented at the annual meeting of the Northern Minnesota Medical Society, Alexandria, Minnesota, September 7, 1956.

Dr. Reichelderfer, formerly Assistant Professor of Pediatrics, University of Minnesota, is now at the Laboratory of Infectious Diseases, National Institutes of Health, Bethesda, Maryland.

Parasites

There are many parasitic agents responsible for diarrhea. The most frequently encountered in children are *Endoamoebia histolytica, Strongyloides, sterocoralis, Giardia lambia* and *Balantidium coli.* All are spread by fecal contamination. These infections are not common in the latitude of Minnesota. They are discussed only for completeness and with the thought that we are living in a world of ever shrinking distances.

The acute phase amoebiasis (*E. histolytica*) requires attention to water loss as in every other diarrhea. Treatment with Terramycin® (oxytetracycline) will destroy large numbers of amoebia and cysts in the colon. Most authorities agree that the broad spectrum antibiotics should be administered with chloroquine and Diodiquin.® Amoebiasis in this part of the world is usually spread by faulty plumbing connections and contaminated water and food.

Strongyloides sterocoralis is found in the soil mostly in warm climates. The worm burrows in the skin of the bare feet, is carried to the lungs and thence coughed up and swallowed into the gastrointestinal tract. There the tiny worms burrow into the crypts of the mucosa of the colon producing necrosis of tissue and a severe diarrhea. Treatment is generally unsatisfactory and the disease becomes chronic. Enteric coated tablets of gentian violet have been used with some success.

Giardia lambia is a flagellate that has been implicated in epidemics of diarrhea in children[3] and young adults. Several of the medical students at the University of Oregon in 1955 were found to have gastroenteritis from this organism. Treatment with atabrine appears to be most efficacious.

Balantidium coli is a ciliate which is usually found in the gastrointestinal tract of swine. Human infections are accidental. Severe diarrhea is produced by this organism. Fortunately, oxytetracycline effectively destroys it.

Fungi

Candida albicans has been implicated in several epidemics of diarrhea of the newborn.[4,5] Diarrhea following widespread use of broad-spectrum antibiotics (allowing C. albicans to replace the normal flora of the gut) has become common in recent years. Infants may be treated wtih gentian violet. Mycostatin® is specific for this organism and should be used whenever possible. Other fungi when ingested with spoiled foods may cause a transitory gastroenteritis.

Bacteria

Staphylococci.—Staphylococcal food poisoning with its resulting gastroenteritis is the result of food being contaminated by a nasal or skin carrier or by open staphylococcal lesions. Frequently the food is allowed to stand several hours before serving during which time the potent enterotoxin is formed. Ham, cream puffs, eclairs and salads frequently are incriminated. Several epidemics have been shown to have their origin in community or church suppers. The onset of the gastroenteritis is sudden, usually within one-half to four hours. Treatment is supportative during the acute phase with emphasis on the replacement of fluids lost in the crampy, frequent stools that are passed. Pasteurization of bakery products, refrigerated storage and elimination of infected food handlers are effective means of prevention.

Severe pseudomembranous enterocolitis from resistant strains of staphylococci may appear as a complication in those patients who have been treated with antibiotics,[6] the staphylococci having replaced the natural flora of the intestines.

A recent epidemic of diarrhea in the newborn nursery of a St. Paul Hospital was shown to be due to Staphylococcus aureus.[7] All affected infants had this organism in their stools and nasopharynx. Several of the nurses harbored this same organism in their nose and throat.

Enteric Bacilli.—The enteric bacteria causing diarrhea in infants and children are those of the following genera: *Vibrio, Shigella, Salmonella, Proteus, Pseudomonas, Paracolon* and *Escherichia.* All are characterized by their inability to ferment lactose in culture media with the exception of the *Escherichia.* The most important members of this group of enteric pathogens in this part of the world are the *Salmonella, Shigella* and the enteropathic coliforms of the genus *Escherichia.* Generally, the drugs that are most effective against this group of organism (in order of their activity)[8] are: Chloromycetin® (chloramphenicol), broad spectrum drugs of the mycin group, sulfadiazine, and neomycin.

Laboratory techniques for the isolation of these organisms have been improved greatly. The use

of selective media such as selinite, desoxycholate and desoxycholate-citrate permits rapid differentiation between pathogens and nonpathogens in the stools. Colonies may be picked from these selective media and placed in sugars for accurate identification. The development of specific sera for agglutination of the enteropathic coliforms and a polyvalent sera for the *Salmonella* and *Shigella* has hastened laboratory diagnosis.

Shigella usually reach the gastrointestinal tract by the media of infected food and water.[2,9,10] The incubation period varies from one to seven days. The pathological process is an inflammatory one which affects the colon and often the terminal ileum. Abdominal cramps and discomfort are present. Blood and mucus are found in the stools. A smear of the mucus, when stained, will show a predominance of polymorphonuclear leukocytes. This will often help to separate a *Shigella* dysentery from a *Salmonella* dysentery before laboratory reports are available. Infants occasionally show meningismus, jitteriness and convulsions.[11] The neurotoxin produced by the *Shigella* produces this effect. The diagnosis of dysentery is often made at the time of a diagnostic lumbar puncture when a foul, loose, bloody, mucous-containing stool is passed as a result of the trauma involved in the procedure. Sulfadiazine and other less soluble sulfa drugs are used to treat shigellosis. Chloromycetin and other mycin drugs may also be used. Polymyxin is effective in clearing up the carrier state. Epidemic diarrhea in nurseries due to this organism is rare.

Salmonella.—The bacteria of the *Salmonella* group include over 150 different strains, the majority of which are animal pathogens.[12] Clinical symptoms may be those of a mild gastroenteritis but more frequently they are typical of an explosive type of food poisoning. Less frequently, the clinical picture is that of a septicemia, or a typhoidal illness.

The old designation of the typhoid organism *Eberthella typhosa* has been changed. It is now grouped with the *Salmonella* and called *S. typhosa.* Typhoid fever is a classic enteric fever. The fever may be accompanied by either diarrhea or constipation after a seven to fourteen-day incubation period. The patient has a generalized septicemia. The organism multiplies in the lymphoid tissue of the small intestines and regional lymph nodes. Intestinal perforation is often a fatal complication. The biliary tract and bone marrow are often infected—thus a chronic carrier state and osteomyelitis may result. Relapses are frequent (10 per cent). Chloromycetin® is the specific antibiotic for the treatment of this condition. Some authorities have used, in addition, ACTH and the other adrenal steroids. The mortality has been reduced by supportative theory and chloromycin.

Enteric fevers caused by *S. paratyphi* A, B and C and *S. shotmulleri* are of shorter duration and often milder than *S. typhosa.* The treatment is the same.

Gastroenteritis produced by the other members of the group of *Salmonella* have a shorter incubation period, eight to forty-eight hours. The patient usually has diarrhea with an accompanying septicemia. The effects of the disease are milder than with either typhoid or paratyphoid fevers. The antibiotic that is most effective in these infections is Chloromycetin. Chronic carriers and subclinical attacks account for the spread of these conditions in humans. *Salmonella* of this group have the species name frequently designating the place where the strain was first isolated, (*S. Montevideo, S. Newport, S. Panama,* etc.) or the name of the animal in which they are most frequently found, (*S. cholerae suis, S. typhimurium, S. gallinarum*).

Contaminated water, shell fish, milk, and community gatherings have been largely responsible for typhoid fever epidemics. Contaminated foods and powdered eggs[13] have accounted for the milder types of salmonellosis. Hardy recently isolated large numbers of *Salmonella* from processed meats of a Florida abattoir.[14]

Pseudomonas and Proteus.—*Proteus* and *Pseudomonas aeruginosa* are enteric bacilli frequently encountered in normal stools. Their sole appearance in diarrheal stools of infants has led many pediatricians to consider them as causative agents of disease.[15] *Proteus morgani* and *P. mirabilis* seem to be found in sick children more frequently than *P. vulgaris.* Replacement of the usual flora of the colon by these antibiotic-resistant organisms has provided a perplexing problem. Occasional strains are sensitive to Chloromycetin and sulfadiazine but most are resistant to all antibiotics. Oral polymyxin and neomycin are often efficacious. Administration by the oral route avoids the nephrotoxic effect of these drugs since they are not absorbed.

Vibrio comma, the causative agent of Asiatic cholera, is not found in the United States. Re-

placement therapy and, to a lesser extent, the antibiotics have robbed this disease of much of its past terror.

Enteropathic Coliforms.—Epidemic diarrhea of the newborn was long thought to be due to a viral agent because no pathogenic organisms could be recovered from the stools. Adam[16] in 1927 found certain fermentative types of *E. coli* associated with diarrhea in infants in Germany. Kauffman[17,18]of Denmark listed an antigenic schema for the *E. coli* group in 1944 and 1947. Bray[19] in 1945 in England using Kauffman's antigenic schema associated a specific type of *E. coli* (O 111:B4) with an epidemic of diarrhea among newborn infants. Findings by several workers[20,22] have shown further that certain serologic strains of *E. coli* could cause gastroenteritis. These bacilli had been overlooked when routine methods of screening stools were used.

Classification is based upon identification of *E. coli* serotypes. Special typing sera is required for their identification. Commercial sera have recently appeared on the open market for this purpose. They are simple to use and can be employed in most laboratories. The 0 antigen or somatic structural antigen is thermostable while the B or K antigen from the envelope or capsule is thermolabile. Identification is based on these properties. There have been twelve serological types and subtypes incriminated[23]: O 111:B4, O 55:B5, O 26:B6, 112a, 1126:B13, O 112a, 112c:B11, O 86:B7, O 119:B14, O 125:B15, O 126:B16 and O 127:B8. Other types will probably be implicated in the future. These enteropathic coliforms were found when several epidemics of diarrhea of the newborn were investigated from stool specimens that had been frozen and stored.[24] Stock and co-workers[25] in the Pittsburgh Children's Hospital showed how an epidemic of twenty-five cases in that hospital spread to five other hospitals and nursing homes in the community and accounted for fifty-one secondary cases.

When epidemic diarrhea is found in a nursery these bacilli and other pathogens must be searched for diligently. The exposed infants should be isolated and observed for at least two weeks. The routines of the nursery should be examined for breakdown of isolation techniques employed by the nurses, formula processing, et cetera. Concurrent disinfection of all discharges and all soiled articles must be undertaken. Terminal sterilization of the equipment and nursery is also required. No new admission should be allowed to the nursery. Known cases should be isolated and treated with neomycin 50 mgm/kilogram body weight and given fluid replacement therapy. The high mortality associated with this disease requires that rigid measures be instituted promptly.

Harris et al[26] isolated enteropathic coliforms from five of thirty-five epidemics of diarrhea of the newborn in New York State in 1946-55. It was of interest that the hospitals with the larger maternity services were involved more frequently than the smaller ones. The case fatality rate for diarrhea from these enteropathic coliforms was 28 per cent.

Viral Agents.—The local physician with his "virus X" and "virus disease" was well aware of the viral nature of diarrhea long before the sophisticated researcher was able to isolate the viruses!

Viral agents as causes of diarrhea have only recently been proved to be important. Diarrhea was known to accompany poliomyelitis and some of the other acute viral infections, but studies in epidemic diarrhea of the newborn for viral agents had been disappointing. Buddingh and Dodd[27] isolated (and were able to keep growing in the cornea of rabbits) a virus that they believed to cause diarrhea in infants. Light and Hodes[28] were able to pass another virus (isolated from an epidemic of diarrhea in newborns) using calves. They showed that the calves had a rise in antibody titre to this agent. Not until Enders and his colleagues[29] developed the methods for isolating virus in tissue culture did the laboratory worker have a tool that enabled him to readily isolate viruses. A few viruses could be grown in chick embryos but this was a limited technique. A large number of new viruses have been isolated from the stools of sick and well children during the past two years by the tissue culture technique of Enders. The exact relationship of many of these viruses to disease is not known.[30,32] Sabin[33] at the Cincinnati Children' Hopital examined the tools of fifty-six children, less than four years of age admitted for gastroenteritis, for pathogens and virus. He was able to recover a viral agent in 43 per cent of the cases. Bacterial pathogens in the seventeen patients from whom viruses were isolated included two *Shigella,* one *Salmonella,* one *Paracolon* and three *Proteus.* Viral isolations from monkey kidney tissue culture gave three poliomye-

litis type 1 viruses, three adenovirus, three Coxsackie, 2 ECHO viruses (Enteric cytopathic human orphan virus) and nine unknown strains. Forty per cent of the children in a Swedish epidemic of Type 3 adenovirus were reported to have had gastroenteritis. It has long been known that the common cold often manifested itself with grippe-like symptoms and gastroenteritis. Many doctors called this intestinal flu. In light of recent discoveries these were probably adenovirus or ECHO viruses.

Other Agents Causing Diarrhea.—Infants that are overfed and receive either too much carbohydrate or protein in the diet will develop diarrhea.[34] Ingested poisons and cathartics will also cause loose, watery stools. These causes can be uncovered by taking a careful history when the child is seen.

Diarrhea that accompanies pellegra is rarely seen in these days of prosperity and improved diets. Nicotinic acid supplementation of refined foods and more-varied diets have practically eliminated this vitamin deficiency state. Tropical sprue with its diarrheal stools has responded to the administration of folic acid.

Coeliac disease and cystic fibrosis of the pancreas (mucovisidosis) often produce loose, watery stools of a chronic nature. The clinician seeing an infant for the first time should keep these conditions in mind. Especially, when after a trial of adequate treatment, the diarrhea continues and no pathogenic agent is found.

Older doctors often attributed diarrhea to inifections elsewhere in the body. These were called parenteral diarrhea, alimentary intoxication or acute nutritional disturbance. Infants often had a myringotomy to relieve their diarrhea.[35] Doubtless, some of these infections did cause loose stools. However, with the use of tissue culture techniques for viral isolation and the discovery of the enteropathic coliforms, many of these uncertain diagnoses will be reported less frequently.

Who cannot recall the diarrhea that accompanies severe states of emotional tension, particularly at examination times. Finally, and not to be forgotten, is the diarrhea that follows the ingestion of certain foods[36]—particularly, that which followed a glorious adventure into a melon patch or orchard where unripe and forbidden fruits were eaten.

Although the causes of diarrhea are varied, the physician, after once establishing that a child has diarrhea, takes steps to identify the organism or to form a hypothesis as to the most likely causative agent. The type of fluid replacement therapy required depends upon the clinical evaluation of the patient—thus treatment is directed towards the patient as well as the cause of his disease.

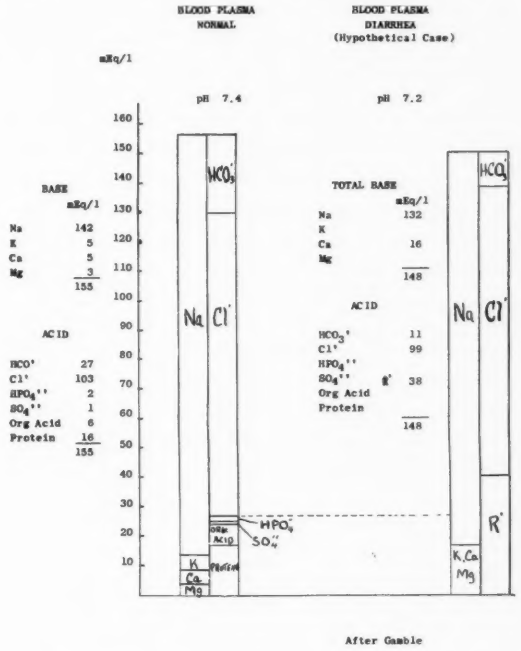


Fig. 1. Comparison of blood plasma determinations between normal and a hypothetical case of diarrhea.

Symptoms

Clinical observations are as important as the result of laboratory studies in the assessment of diarrhea. In infants there is loss of weight, the skin loses its elasticity and may be pulled up in folds, the eyes and fontanelles are sunken and oliguria may be present. These are all signs of depletion of interstitial fluid and plasma volume. As the diarrhea progresses, circulatory insufficiency develops, the peripheral pulses become feeble, the extremities become cold and ashen and the rate of the heart is increased. The infant is restless and irritable. The child, as circulatory collapse progresses, becomes comatose and may appear cyanotic or pallid. It is remarkable to see these signs disappear when adequate fluid replacement therapy is given.

The diseases causing diarrhea contribute impairment of absorption of water and solutes from the gut, and the subsequent loss of additional water and electrolyte in the stools. The ions lost in the stool include sodium and chloride from the extracellular fluid and potassium, magnesium, calcium and phosphorus from the cells and bone. Infants in the early stages of diarrhea often maintain their nutritive, water and electrolyte balance as long as they continue to eat and drink. Refusal to feed and supervening vomiting occur as the diarrhea progresses. The children are then unable to keep up and replace the loss of gastrointestinal secretions lost in the stools. The additional effects of infection and fever cause depletion of liver glycogen and breakdown of cell protein. Loss of interstitial fluid and plasma volume causes further impairment of the circulation, diminution of renal flow and oliguria. These changes are represented after the manner of Gamble in Fig. 1.[37] The electrolyte and water regulating mechanism of the kidney is lost with the retention of the products of cell catabolism. Alteration in the composition of the body fluids may be reflected in the renal tubules, central nervous system and cardiac muscle. The usual finding in diarrhea is that of a metabolic acidosis due to the greater loss of sodium than chlorides in the stool and the reduction in plasma volume. Water loss may be disproportionate to electrolyte loss and a high serum sodium may be seen in some cases. Alkalosis is rare in diarrhea but has been reported by two observers.[38,39]

Treatment

The method of treatment for diarrhea varies from one physician to another. All, generally follow the comprehensive and systematic plans of Park,[40] Powers,[41] Darrow,[42] Hartman, [35] Butler and Talbot,[43] Gamble,[44] Dodd and Rapoport[45] and Harrison.[46] The emphasis is upon, "the administration of fluids, the transfusion of blood, the withholding of food for a period of time and the administration of food at the end of starvation in gradually increasing amounts."[41] Present-day techniques of fluid administration and electrolyte replacement have greatly simplified therapy. Harrison's method[46] of treatment, because of its ease of adaptibility and simplicity, is recommended for the practicing doctor.

The infant with mild diarrhea from infection may require only simple management at home. Antibiotics, withholding food, and the feeding of an oral solution made of one and a half tablespoons of sugar and half teaspoon of salt to one quart of water may be all that is required. The more colorable physician may prefer to use a commercially-available solution (Lytren) for the oral solution to replace electrolyte. The withholding of solid foods lessen the abdominal distention and decreases the volume of the diarrheal stools. Feedings are gradually resumed over a period of several days. Usually the infant may be put back to breast or started on skim milk feedings when the number of stools has decreased. Over a period of four to five days, the infant is gradually restored to his full caloric requirements.

Chung[47,48] has questioned the advantage of withholding food. He showed in a small series of patients with diarrhea that the intestinal absorption of nitrogen, fat, sodium, calcium, potassium and chloride was improved on a high dietary intake even though the amount of loss in the stools was increased. He compared the clinical courses of infants with diarrhea on "starvation" and "full feeding" regimes and, obtained a more favorable response in those infants on the full-feeding regime. Most clinicians still adhere to Power's method of withholding food.

Opiates and bismuth preparations have little benefit in children on the course of loose stools. Pectin and kaolin may thicken the stool for asthetic purposes. The use of cathartics only further irritates the bowel and aggravates water loss. It is well to keep in mind that the fluid and electrolyte replacement therapy is the most important part of treatment and that little is gained by these additional measures.

The generalist treating mild diarrhea can undertake replacement therapy with meager clinical information. Severe diarrhea that is accompanied by anorexia, vomiting, severe dehydration, circulatory collapse, renal failure, and shock requires, in addition to the data obtained by physical examination and history, the support of a clinical laboratory. An infant with severe diarrhea should be hospitalized. The following laboratory determinations should be performed on admission: Complete blood count (including hemoglobin and hematocrit), CO_2, chloride, sodium, potassium, pH and NPN determinations of the blood. The physician, smaller hospital or clinic—unable to afford the luxury of a clinical laboratory—may resort to Scribner's[49,50] methods for bedside deter-

ORAL ELECTROLYTE SOLUTION (LYTREN)*

Composition	mEq/L			
KCl, NaCl, Na Citrate	Na	50	Citrate	30
NaH_2PO_4, Ca Lactate	K	20	SO_4	4
$MgSO_4$ and Dextri-Maltose	Ca	4	Lactate	4
	Mg	4	PO_4	10
			Chloride	30

10 gm. to 120 cc. water
Mead Johnson and Company

minations of CO_2 and chlorides. *The main value of chemical determinations is to serve as a support of clinical judgment.* Accurate records of fluid administration should be kept day by day, and checks made of the serum electrolytes to support the clinical status of the patient. The simple measurement of body weight supplies an excellent index of the degree of rehydration.

The infant suffering from the effects of severe dehydration and electrolyte loss should receive immediate emergency treatment by parenteral routes. Intravenous infusions of 10 per cent glucose, 20 cc/kilogram body weight and plasma or whole blood in the same amount, are used. These increase circulating blood volume, interstitial fluids, provide water so that circulation, renal blood flow and urine output will be improved, and replace liver glycogen. This is followed by an infusion of a mixture of Lactate-Ringer's solution† and 5 per cent glucose solution in equal parts—80 cc/kilogram body weight. Slow subcutaneous infusion at the rate of 0.5-1.0 cc/min is satisfactory for most infants. This phase of treatment is usually accomplished during the first twelve hours.

Infants may be considered improved if skin turgor is more normal, they are more reactive and alert, voiding frequently and have good peripheral circulation. They are usually thirsty, and oral feedings of electrolyte and carbohydrate solutions (Lytren)** may be begun despite the continual stooling. The oral electrolyte solutions are given six to eight times a day in the amount of 150 cc. or more per kilogram body weight. This solution contains sodium, potassium and chloride. There is no need for potassium replacement in the acute phase of the diarrhea. This solution will provide 3 mEq of potassium per kilogram of body weight per day. This is sufficient for replacement even though some is lost through the stools. Oral feedings of electrolyte solutions are usually continued for two to three days and milk feedings gradually introduced until maintenance calories are provided. Infants that are anemic and malnourished may require transfusions and a high-caloric balanced diet after they have responded to the initial phase of treatment.

When the etiologic agent of the diarrhea is known, the appropriate antibiotic is given with the replacement therapy. When it is unknown, Chloromycin and sulfadiazine or the other broad-spectrum antibiotics, seem to be the drugs of choice. Vitamin C and the soluble vitamins of the B-complex may be given parenterally in severely ill children.

RINGER'S LACTATE SOLUTION (HARTMANN)*

	gm/l		mEq/l		
Na lactate	3.1	Na	130	Cl	109
NaCi	6.0	K	4	Lactate	28*
K Cl	0.4	Ca	3		
Ca Cl_2	.2				

The majority of children will respond well to the above phase of management. There are some infants who have a disproportionate loss of water to sodium from hyperventilation, fever and diarrhea. This state has been termed hyperosmolarity.[51,52] Central nervous system signs and convulsions are seen in this group. The skin has a firm velvety resistant texture. Treatment is modified to give more glucose, water and solutions containing less sodium. When a child has an extreme degree of sodium depletion as evidenced by marked loss of interstitial fluid and circulatory collapse and, if the determinations of serum show a lowered sodium (or $CO_2 + Cl + 10$). Ringer's lactate should be administered without the addition of 5 per cent glucose. No specific treatment for the metabolic acidosis is undertaken and the full advantage is taken of the regulatory power of the functioning kidney. The extracellular fluid is expanded by Lactate Ringer's solution and 5 per cent glucose water which contains sodium ions in excess of chlorine. The water intake is adequate so that diuresis occurs and the acidosis is gradually corrected.[53]

Harrison has shown after six years of experience that, "when laboratory data cannot be obtained rapidly and the clinical appraisal does not suggest usual sodium depletion or disproportionate water deficit this program of therapy is one of choice."[46] The author has used it with minor variations during the past four years and found that he is

*Metabolized to HCO_3
†See Ringer-Lactate Formula
**Lytren Formula

in agreement with these findings, and that with this system the minimal use of the laboratory has been required.

Children above the age of two years seldom require the intensive treatment of diarrhea as outlined above.[54] They are better able to maintain their homeostasis, despite their frequent loose stools. Usually an adequate fluid intake plus treatment with antibiotics is all that is required.

Summary

The primary purpose of presenting a paper to an assembly such as this is to further postgraduate medical education. The author has endeavored to use good pedagogic procedure in relating the well-known causes of diarrhea to those recently reported as causing diarrhea in children. To this end, the field of acute diarrhea as a manifestation of disease and the causative agents of acute diarrhea have been reviewed. Treatment of the acute diarrheal state has been outlined.

References

1. Baker, J. A., Neter, E., et al: Epidemic and endemic diarrheal diseases of the infant. Ann. New York Acad. Sc., 66:3-230, 1956.
2. Stewart, W. H., Hardy, A. V., and Watt, J.: Shigellosis (bacillary dysentary). *In* Brennerman, J. (Ed.): Practice of Pediatrics. Vol. II, Chapt. 5. Hagerstown, Md.: W. F. Prior Co., Inc., 1948.
3. Veghelyi, P.: Giardiasis. Am. J. Dis. Child, 59: 793, 1940.
4. Durand, J. I.: Epidemic diarrhea in a hospital apparently caused by monilium. J. Pediat., 7:726, 1935.
5. Murphy, J. R., and Mallozzi, M.: Fungus findings in a diarrheal outbreak in newborns. Arch. Pediat., 53:276, 1936.
6. Williams, E.: Staphylococcic pseudomembranous enterocolitis complicating treatment with Aureomycin. Lancet 2:999, 1954.
7. Hedenstrom, F.: Personal Communication.
8. Antibiotics Clinical Trials Committee, Medical Research Council: Antibiotics and chemotherapeutic agents in treatment of infantile diarrhea and vomiting. Lancet 2:1163, 1953.
9. Higgins, A. R., Floyd, T. M., and Kader, M. A.: Studies in shigellosis II. Observations in incidence and etiology of diarrheal disease in Egyptian village children. Am. J. Trop. Med., 4:271, 1955.
10. Floyd, T. M., Higgins, A. R., and Kader, M. A.: Studies in shigellosis V. The relationship of age to incidence of shigella infections in Egyptian children with special reference to shigellosis in the newborn and in infants in the first six months of life. Am. J. Trop. Med., 5:119, 1956.
11. Donald, W. D., Winkler, C. H., and Bargeron, L. M., Jr.: The occurrence of convulsions in children with shigella gastroenteritis. J. Pediat., 48:323, 1956.
12. Schwentker, F. F.: Salmonella infections. *In* Holt's Pediatrics. 12th Ed. New York: Appleton-Century Crofts, Inc., 1955.
13. Abramson, A., Greenberg, M., Plotkin, S., and Oldenbusch, C.: Food poisoning in infants caused by egg yolk powder. Am. J. Dis. Child., 87:1, 1954.
14. Galton, M. M., Mackel, D. C., Lewis, A. L., Haire, W. C., and Hardy, A. V.: Salmonellosis in poultry and poultry processing plants in Florida. Am. J. Vet. Res., 16:132, 1955.
15. Graber, C. C., and Dodd, M. C.: The role of *paracolobactrum* and *proteus* in infantile diarrhea. Ann. New York Acad. Sc., 66:136, 1956.
16. Adam, A.: Dyspepsiekoli zur frage der bakteriellen, Aetioligie der sogenannten alimentären intoxication. Jahrb. Kinderheilk., 116:8, 1927.
17. Kauffmann, F.: Zur serologie de *Coli gruppe*. Acta path. et microbiol. scandinav., 21:20, 1944.
18. Kauffmann, F.: Serology of the coli group. J. Immunol., 57:71, 1947.
19. Bray, J.: Isolation of antigenically homologenous strains of *Bact. coli neopolitanum* from summer diarrhea of infants. J. Path. & Bact., 57:239, 1945.
20. Ferguson, W. W., and June, R. C.: Experiments in feeding volunteers with *E. coli* 0111:B4, a coliform organism associated with infant diarrhea. Am. J. Hyg., 55:155, 1952.
21. Stulberg, C. S., Zuelzer, W. W., and Nolke, A. C.: An epidemic of diarrhea of the newborn caused by *E. coli* 0111:B4, Pediat., 14:133, 1954.
22. Wheeler, W. E., and Wainerman, B.: The treatment and prevention of epidemic diarrhea due to *E. coli* 0111 by the use of chloramphenicol and neomycin. Pediat., 14:357, 1954.
23. Report of Committee on Control of Infectious Diseases: American Academy of Pediatrics, page 21, 1955.
24. Neter, E., Korns, R. F., and Trussell, R. E.: Association of *E. coli* sero group 0111 with two hospital outbreaks of diarrhea of the newborn infant in New York State in 1947. Pediat., 12:377, 1953.
25. Stock, A. H., and Shuman, M. E.: Gastroenteritis in infants associated with specific serotypes of *E. coli* II. Pediat., 17:196, 1956.
26. Harris, A. H., Yankauer, A., Greene, D. C., Coleman, M. B., and Phaneuf, M. Y.: Control of epidemic diarrhea of the newborn in hospital nurseries and pediatric wards. Ann. New York Acad. Sc., 66:118, 1956.
27. Buddingh, G. J., and Dodd, K.: Stomatitis and diarrhea of infants caused by a hitherto unrecognized virus. J. Pediat., 24:105, 1944.
28. Light, J. S., and Hodes, H. L.: Isolation of a filtrable agent causing diarrhea in calves. Am. J. Pub. Health, 33:1451, 1943.
29. Enders, J. F., Wheeler, T. H., and Robbins, F. C.: Cultivation of the Lansing strain of poliomyelitis virus in cultures of various human embryonic tissues. Science, 109:85, 1949.
30. Ramos, Alvarez, M., and Sabin, A. B.: Intestinal viral flora of healthy children demonstrated by monkey kidney tissue culture. Am. J. Pub. Health, 46:295, 1956.
31. Honig, E. L., Melnick, J. L., Isacson, P., Parr, R., Myers, I., and Walton, Mary: An epidemological study of enteric virus infections. J. Exper. Med., 103:247, 1956.
32. Blattner, R. J.: Enteric viruses. J. Pediat., 48: 682, 1956.
33. Sabin, A. B.: The significance of viruses recovered from the intestinal tracts of healthy infants and children. Ann. New York Acad. Sc., 66:226, 1956.
34. Anderson, J. A.: Minor nutritional and digestive disorders of the artificially fed infant. *In* Brennerman, J. (Ed.): Practice of Pediatrics. Vol. I, Chapt. 26. Hagerstown, Md.: W. F. Prior Co., Inc., 1948.
35. Hartmann, A. F.: Chemical changes occurring as the result of certain diseases. Effects of diarrhea, vomiting and oliguria on acid base balance of plasma of infants with mastoiditis. Am. J. Dis. Child., 35:557, 1928.

36. Karelitz, S.: Diarrhea in infants and children, Pediat. Clin. North America, 137, (February) 1956.
37. Gamble, J. L.: Chemical anatomy, physiology and pathology of extracellular fluid. Cambridge: Harvard University Press, 1951.
38. Gamble, J. L., Fakey, K. E., Appleton, J., and MacLachlan, E.: Congenital alkalosis with diarrhea. J. Pediat., 26:509, 1945.
39. Darrow, D. C.: Congenital alkalosis with diarrhea. J. Pediat., 26:519, 1945.
40. Park, E. A.: Le traitement de la maladie dite, Intoxication alimentaire Re franc de pediat. Page 108, 1925.
41. Powers, G. G.: A comprehensive plan of treatment for so called intestinal intoxication of infants. Am. J. Dis. Child., 32:232, 1926.
42. Darrow, D. C., Pratt, E. L., Flett, J., Gamble, A. H., and Wiese, H. F.: Disturbances of water and electrolytes in infantile diarrhea. Pediat., 3:129, 1949.
43. Butler, A. M., and Talbot, N. B.: Parenteral fluid therapy I, Estimation and provision of daily maintenance requirements. II, Estimation of losses incident to stravation and dehydration with acidosis or alkalosis and the provision of repair therapy. New Eng. J. Med., 231:585-590, 621-628, 1944.
44. Gamble, J. L.: Deficits in diarrhea. J. Pediat., 30:488, 1947.
45. Dodd, K., and Rapoport, S.: Fluid therapy in acute illness of infancy and childhood. Cincinnati J. Med., 30:135, 1949.
46. Harrison, H. E.: The treatment of diarrhea in infancy. Pediat. Clin. North America, 335 (May) 1954.
47. Chung, A. W.: The effect of oral feeding at different levels on the absorption of foodstuffs in infantile diarrhea. J. Pediat., 33:1, 1948.
48. Chung, A. W., and Viscorova, B.: The effect of early oral feeding versus early oral stravation on the course of infantile diarrhea. J. Pediat., 33:14, 1948.
49. Scribner, B. H.: Bedside determination of chloride: A method for plasma, urine and other fluids and its application to fluid balance problems. Proc. Mayo Clinic, 25:209, 1950.
50. Scribner, B. H.: The bedside determination of bicarbonate. Proc. Staff Meet. Mayo Clinic, 25:641, 1950.
51. Finberg, L., and Harrison, H. E.: Hypernatremia in infants. Pediat., 16:1, 1955.
52. Weil, W. B., and Wallace, W. M.: Hypertonic dehydration in infancy. Pediat., 17:171, 1956.
53. Cheek, D. B.: Changes in total chloride and acid base balance in gastroenteritis following treatment with large and small loads of sodium chloride. Pediat., 17:839, 1956.
54. Wolfish, M. G.: Acute gastroenteritis. J. Pediat., 43:675, 1953.

"MISSILE AND SATELLITE EMPHASIS MUST NOT HOLD BACK MEDICAL RESEARCH"

A warning that the current preoccupation with missiles and satellites must not be permitted to hold back vital life-saving research in the medical sciences was sounded by Dr. Robert W. Wilkins of Boston, President of the American Heart Association, speaking before a luncheon meeting of the Advertising Club of Baltimore, held in January as a forerunner of the 1958 Heart Fund campaign being conducted during the entire month of February.

In his talk, Dr. Wilkins urged that "wholehearted support, not just lip service, be given to the biological sciences and the needs for research and education in this area so that we may break through the barriers of ignorance in our struggle against disease. In our preoccupation with military security and the physical sciences, we must not allow the medical scientist to assume the status of a forgotten man."

Dr. Wilkins said that basic research, which he defined as the search for knowledge without commitment to immediate application or cures, "holds the key to further advances in all fields." To the extent that basic research is adequately understood and supported, he said, "we shall go forward to ever greater mastery of the world in which we live," a mastery that includes the conquest of disease.

"Let me make it perfectly clear," Dr. Wilkins told his audience of advertising men, "that I join you, and all other good Americans, in the fervent prayer that our scientists will achieve whatever is necessary in order to protect the security of our nation and our way of life. But at the same time," he added, "I should like to express the hope that we do not permit our perspective to become blurred," with regard to the necessity of supporting the biological sciences.

Somewhere in the United States at this very moment, Dr. Wilkin said, there may be an "underpaid and unsung medical scientist" who may in time make as valid a claim to enduring fame as the developers of satellites and missiles by finding the exact causes for high blood pressure and hardening of the arteries. These disorders are responsible for about 90 per cent of all heart disease.

"The achievements of this medical scientist," Dr. Wilkins said, "will be reflected in the saving of millions of human lives, rather than in the perfection of methods of mass destruction. Since the diseases of the heart and blood vessels are now responsible for more than 800,000 deaths yearly in this country alone, or about 54 per cent of all deaths, the unidentified scientist of whom I speak will have done as much, if not more, to advance and benefit humanity than any of the nuclear or missile scientists."

In general, however, Dr. Wilkins said there recently have been signs that the "so-called awakening" to the true meaning of basic research is more apparent than real. "There are evidences that the tributes we hear are indeed mere lip service, based on inadequate comprehension of what basic research really means," Dr. Wilkins noted, and he added, "If the process of investigating *all* aspects of our world, through the physical and biological sciences, is to be held back by an insistence on immediate results in the arms race, we may again find ourselves back in the same boat. We shall be seriously behind in a field on which the welfare and survival of our people depend. This can be prevented if we truly acknowledge the scope of basic research and give it the financial encouragement it needs."

The Current Status of Hereditary Pancreatitis

JOHN B. GROSS, M.D.
MANDRED W. COMFORT, M.D.
JOHN A. ULRICH, Ph.D.
Rochester, Minnesota

IN 1952 Comfort and Steinberg[1] reported the unusual occurrence of pancreatitis affecting four members of a single family and possibly affecting two others. At that time such familial incidence was unique, although Collett and Kennedy[2] in 1948 had reported the occurrence of chronic relapsing pancreatitis in a child with possible involvement of a sibling, and two years later Poulsen[3] had published a similar observation. In each of these two latter reports the child affected with pancreatitis and the sibling suspected of having the disease, exhibited hyperlipemia. Earlier this year we[4] reported the pedigrees and additional observations based on study of two other families with hereditary pancreatitis. In one of these families seven persons in three generations are affected, and pancreatitis is rather strongly suspected in seven of their blood relatives; in addition, another eight persons in this family have experienced atypical abdominal pains of some severity. Jackson[5] recently has encountered a family in which two brothers definitely have pancreatitis and their mother and a child of one of the brothers may be affected also; in this family hyperparathyroidism also prevails. In the past two years we have encountered at the Mayo Clinic several other families in which two or more members definitely have had pancreatitis.

Clinical and Genetic Aspects

What, then, are the clinical features of hereditary pancreatitis, and how does this form of the disease differ from nonhereditary pancreatitis? It should be noted at the outset that experience with hereditary pancreatitis is still small, so that any generalizations are necessarily of a rather tentative nature. Our experience to date indicates that hereditary pancreatitis resembles in most ways the nonhereditary form of the disease.[1,4,6,7] Hereditary pancreatitis is characterized by the same severe, prolonged seizures of pain in the upper abdomen and back, and in the course of time the resultant pancreatic destruction may be followed by any or all of the same sequelae: diabetes mellitus, pancreatic calcifications, external pancreatic insufficiency with steatorrhea and azotorrhea, and formation of pseudocysts.

Differentiation from Nonhereditary Pancreatitis.—Nevertheless, there appear to be certain differences between the hereditary and nonhereditary forms of pancreatitis. The first, of course, is the familial incidence in the hereditary form; not only are multiple members of a kindred affected, but such involvement seems to be in accordance with Mendelian laws in these families. A second, striking difference between the two forms of the disease is the onset early in life in hereditary pancreatitis. Whereas in the nonhereditary form of the disease the attacks usually begin in the fourth decade of life,[6,8] in most cases of hereditary pancreatitis the attacks begin in childhood. In our experience this onset early in life has been a very helpful diagnostic feature, so that we have learned to suspect rather strongly the hereditary form of pancreatitis when the patient is a child or young adult, or when the attacks date back to childhood. Under these circumstances a painstaking inquiry into the familial history is likely to be rewarded with the discovery of other such cases.

A few other clinical differences between the hereditary and the nonhereditary forms of pancreatitis deserve mention. So far, hereditary pancreatitis has affected females approximately one and one-half times as frequently as males, whereas in most reported series of nonhereditary pancreatitis males have outnumbered females by two or three to one, and in one series by six to one.[6,8] This apparent slight predilection of hereditary pancreatitis for the female may, however, reflect

Dr. Gross is from the Section of Medicine, Mayo Clinic and Mayo Foundation, Rochester, Minnesota, as was Dr. Comfort until his death August 7, 1957. Dr. Ulrich is in the Section of Biochemistry. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Read at the meeting of the Minnesota Society of Internal Medicine, Rochester, Minnesota, October 21, 1957.

only chance variation in an admittedly small sample. It is interesting that among the cases of hereditary pancreatitis there has been none so far with associated gallstones or disease of the biliary tract. Also of interest, as was observed in the family reported by Comfort and Steinberg,[1] is the fact that the pancreatic calcifications encountered among cases of hereditary pancreatitis have been almost exclusively calculi in the larger pancreatic ducts. Hyperlipemia has been observed only once, and alcoholism has been conspicuously absent from cases of hereditary pancreatitis, although a few of the patients stated that they had formerly used alcohol but had for some time abstained completely, since alcohol had precipitated attacks of abdominal pain.

Although hereditary pancreatitis has been recognized rather infrequently to date, it seems likely that cases will be encountered more frequently as acquaintance with the entity increases. It has been instructive to note the frequency with which bouts of pancreatitis have masqueraded as duodenal ulcer, gastritis, gallstone colic, and other similar entities in the hereditary cases.

Heritability.—As yet, the genetic observations are somewhat tentative also, and are based partly on the presumption that most of those persons in the affected families now only suspected of having had attacks of pancreatitis eventually will prove to have the disease. Such a presumption appears not unjustified, since the description of the attacks of abdominal pain in most of these persons is characteristic of pancreatitis. The available evidence indicates that the mechanism of inheritance is a nonsex-linked Mendelian dominant gene.[1,4] It is not yet settled whether the phenomenon of poor penetrance sometimes is involved; in such instances it is postulated that the person in question has inherited the abnormal trait, perhaps in heterozygous fashion, although without development of the clinical manifestations of the disease.

Etiologic and Pathogenic Considerations

The etiology and pathogenesis of chronic relapsing pancreatitis are unknown. In hereditary pancreatitis Nature seems to have provided us with an unusual opportunity and challenge, for the development of the disease in accordance with Mendelian laws in some families and the appearance early in life in the person affected make it appear virtually certain that they have inherited some predisposing abnormality. Although the evidence now available is rather meager, it seems that such an inherited abnormality is more likely metabolic or biochemical than anatomic in nature.

In an effort to obtain evidence which might bear on the nature of the presumed inherited defect predisposing to hereditary pancreatitis, we[9] assayed, microbiologically, the apparent free and total (that is, free and combined) forms of fourteen individual amino acids in fasting serum and twenty-four-hour urine specimens from two groups of persons: in the first group were seven persons with hereditary pancreatitis and four of their blood relatives apparently without the disease; in the second group were eleven persons with the nonhereditary form of pancreatitis.

Amino Acid Abnormalities.—Certain patterns of the serum and urinary amino acids observed in both groups with pancreatitis appeared abnormal when the data were compared with those obtained previously[9-11] in study of healthy persons. Further, the patterns of abnormality were somewhat different for the hereditary and the nonhereditary groups. The most distinctive and outstanding abnormality encountered in the patients with hereditary pancreatitis was excessive urinary excretion of lysine. Excessive excretion of leucine occurred in both groups. This amino-aciduria (lysinuria) was observed in multiple members and generations of several families with hereditary pancreatitis, and in one seemingly healthy blood relative in each of three such families (two of these relatives being children). It is of particular interest that the phenomenon of lysinuria was largely confined to the group with hereditary pancreatitis. The amino-aciduria did not seem to be related to the state of activity of the pancreatitis when such existed or to the presence or absence of diabetes mellitus.

Quantitatively, the urinary losses of amino acids were comparable to those which have been found by others in cases of diabetes mellitus, pneumonia, and hepatic disease.[12-15] The largest excretion of lysine was observed in one case of hereditary pancreatitis with general amino-aciduria; in this case the losses were approximately 25 per cent as large as those which have been described in cases of cystinuria, hepatolenticular degeneration, and Fanconi's syndrome.[14-31]

Lysinuria has been described as part of a more or less general amino-aciduria in a number of

conditions, including Wilson's disease and Fanconi's syndrome, and is also an integral part of the more limited amino-acidurias of cystinuria and pernicious anemia in relapse.[18,21-23,29-38] However, in these other conditions, except for cystinuria, in which the urinary excretion of lysine commonly exceeds that of cystine,[29] the excessive urinary excretion of lysine is not the major amino acid defect observed, as it appears to be in hereditary pancreatitis.

The pathogenesis of the observed abnormalities of amino acids in serum and urine of persons with pancreatitis is not clear. The data do not justify exclusion from consideration of either the overflow mechanism or impairment of renal tubular reabsorption as possible factors in the pathogenesis of the observed amino-aciduria; it may be that both factors play some role. It is possible that the seemingly distinctive amino-aciduria in the cases of hereditary pancreatitis reflects an inborn error of protein metabolism, as occurs in phenylketonuria[38-42] and perhaps in Wilson's disease.[25,26,43,44] It is to be hoped that renal clearance studies of lysine and leucine in these cases may provide information which will serve to answer some of the still unanswered questions.

Porphyrin Abnormalities.—Brief mention may be made of certain other observations which also, unfortunately, at present serve only to raise more perplexing questions. In one of the families with hereditary pancreatitis the authors[4] encountered weakly positive results for porphobilinogen on qualitative testing of urine of one man with definite hereditary pancreatitis and sequelae and that of two of his nephews, one of whom had experienced attacks of abdominal pain characteristic of pancreatitis. Both nephews (brothers) exhibited the unusual amino-aciduria characterized by excessive excretion of lysine and, in fact, had almost identical patterns of amino acids in serum and urine. Again, in the case of a young man of another family with hereditary pancreatitis a weakly positive qualitative result for urinary porphobilinogen was encountered. This person had experienced classic attacks of pancreatitis over a period of ten years and had passed red urine on one occasion several years prior to the study. Analysis of his feces disclosed slightly elevated values for fecal coproporphyrin and protoporphyrin. One hesitates to attach much importance to the borderline porphyrin abnormalities observed; and even if these should prove significant, any possible relationship between such porphyrin abnormalities and the pancreatitis, or between these abnormalities and the amino acid abnormalities observed, remains conjectural at present.

It is of interest in this regard, however, to note that Berger and Goldberg[45] recently have reported the association of amino-aciduria and excessive excretion of coproporphyrin III and some intermediate porphyrins in the stool and urine of a boy, both his parents, and a paternal aunt, and have termed this condition hereditary coproporphyria. The amino-aciduria in these cases was characterized by what was described as a "superglycine pattern."

The association of amino-aciduria and porphyrin abnormalities was also described last year by Baron and associates,[33,46] in what they have designated as "H, or Hartnup, disease." In this interesting syndrome, which also seems to be inherited, four siblings of eight from a marriage of first cousins exhibited a tendency to develop a pellagra-like rash on exposure to sunlight, sometimes associated with severe cerebellar ataxia and mental retardation. The constant amino-aciduria in Baron's cases appeared to be of renal origin and of a unique pattern, which included moderately increased excretion of lysine as determined chromatographically. The feces of these persons were found to contain moderately increased quantities of protoporphyrin.

Whether the association of abnormal excretion of porphyrins and amino acids observed to date is more than coincidental remains to be determined. It may be appropriate here to mention also the two cases reported by Saint and associates[47] in which porphyria and pancreatitis coexisted; these authors suggested that the pancreatitis had resulted from focal ischemia as a consequence of porphyric involvement of the smooth muscle in the arterioles of the pancreas.

Association with Hyperparathyroidism.—The interesting family described by Jackson,[5] in which parathyroid adenomas and hyperparathyroidism had developed in multiple members, two of whom were said to have experienced recurrent bouts of pancreatitis, brings to mind the possibility of some inherited defect having to do with calcium metabolism in those who exhibit the hereditary form of pancreatitis. In recent years there have been

sporadic reports of pancreatitis and hyperparathyroidism coexistent in the same person, and the number of such reports seems to be growing.[48-52] In such cases it usually has been postulated that the hyperparathyroidism has resulted in the formation of stones in the pancreatic ducts, with the development of pancreatitis secondary to this ductal obstruction. In our own cases of hereditary pancreatitis the evidence to date seems to point away from the possibility that the pancreatitis was secondary to hyperparathyroidism. The values for serum calcium obtained from some of these persons between attacks have not been elevated; and in a number of instances the pancreatitis has been unassociated with pancreatic calcifications, or has preceded the development of such calcifications by some years.

Treatment

In the present state of our knowledge the management of cases of hereditary pancreatitis is the same as for cases of nonhereditary pancreatitis. Prophylactically, the physician has little to offer such patients except avoidance of alcohol and spices. Nonsurgical management otherwise consists of supportive measures and replacement therapy. The surgical approach, although still leaving much to be desired, has more to offer such patients but must be individualized. In general, it has seemed best to avoid operating on the children and young adults who have hereditary pancreatitis until the trend, tempo, and severity of the disease make some form of surgical intervention seem clearly advisable.

Summary

In some families, pancreatitis seems to be inherited via a Mendelian dominant gene, nonsex-linked. Hereditary pancreatitis almost always begins in childhood or early adult life. It is characterized by the same severe, prolonged seizures of abdominal pain as mark the nonhereditary form. To date, hereditary pancreatitis has been found to occur somewhat more frequently in females and has been unassociated with gallstones, hyperlipemia (one case excepted) or alcoholism.

It appears likely that persons with hereditary pancreatitis have inherited some predisposing abnormality, perhaps of a metabolic nature. Microbiologic assays have revealed certain abnormalities of amino acids in the serum and urine of persons with hereditary pancreatitis and in a few instances in their blood relatives not affected with pancreatitis thus far; these amino acid abnormalities are somewhat different from those observed in persons with nonhereditary pancreatitis. The amino-aciduria of hereditary pancreatitis is rather distinctive in that there is excessive excretion of lysine. The observed abnormalities of amino acids may signify some defect in intermediary protein metabolism. Whether the amino-aciduria may stem, at least partly, from impairment of renal tubular reabsorption is not yet known. Borderline porphyrin abnormalities have been encountered in a few persons with hereditary pancreatitis and in one blood relative without pancreatitis; the significance of this observation, if any, is not now apparent. To date none of the cases of hereditary pancreatitis observed by the authors has been found to have coexistent hyperparathyroidism. The treatment of hereditary pancreatitis at present is the same as that for the nonhereditary form of the disease.

References

1. Comfort, M. W., and Steinberg, A. G.: Pedigree of a family with hereditary chronic relapsing pancreatitis. Gastroenterology, 21:54-63 (May) 1952.
2. Collett, R. W., and Kennedy, R. L. J.: Chronic relapsing pancreatitis associated with hyperlipemia in an eight-year-old boy. Proc. Staff Meet., Mayo Clin., 23:158-162 (Mar. 31) 1948.
3. Poulsen, H. M.: Familial Lipaemia: A new form of lipoidosis showing increase in neutral fats combined with attacks of acute pancreatitis. Acta med. Scanindav., 138:413-420, 1950.
4. Gross, J. B., and Comfort, M. W.: Hereditary pancreatitis: Report on two additional families. Gastroenterology, 32:829-854 (May) 1957.
5. Jackson, C. E.: Hereditary hyperparathyroidism associated with recurrent pancreatitis. (Abstr.) Clin. Res. Proc., 5:185 (Apr.) 1957.
6. Comfort, M. W.; Gambill, E. E., and Baggenstoss, A. H.: Chronic relapsing pancreatitis: A study of twenty-nine cases without associated disease of the biliary or gastro-intestinal tract. Gastroenterology, 6:239-285 (Apr.); 376-408 (May) 1946.
7. Gross, J. B., and Comfort, M. W.: Chronic pancreatitis. Am. J. Med., 21:596-617 (Oct.) 1956.
8. Maimon, S. N.; Kirsner, J. B., and Palmer, W. L.: Chronic recurrent pancreatitis: A clinical study of twenty cases. Arch. Int. Med., 81:56-72 (Jan.) 1948.
9. Gross, J. B.; Comfort, M. W., and Ulrich, J. A.: Abnormalities of serum and urinary amino acids in hereditary and nonhereditary pancreatitis. Tr. A. Am. Physicians, 70:127-138, 1957.
10. Ulrich, J. A.: Urinary excretion of amino acids by human subjects on unrestricted diets. Proc. Staff Meet., Mayo Clin., 29:210-214 (Apr. 21) 1954.
11. Ulrich, J. A.: Microbiologic assays of 14 individual amino acids in the serum of normal persons. (Unpublished data.)
12. Frankl, Willi; Martin, Helen, and Dunn, M. S.: The apparent concentration of free tryptophan, histidine and cystine in pathological human urine measured microbiologically. Arch. Biochem., 13:103-112 (Apr.) 1947.

13. Gabuzda, G. J., Jr.; Eckhardt, R. D., and Davidson, C. S.: Urinary excretion of amino acids in patients with cirrhosis of the liver and in normal adults. J. Clin. Investigation, 31:1015-1022 (Nov.) 1952.
14. Eckhardt, R. D.; Cooper, A. M.; Faloon, W. W., and Davidson, C. S.: The urinary excretion of amino acids in man. Tr. New York Acad. Sc. s. 2., 10:284-290 (June) 1948.
15. Walshe, J. M.: Disturbances of amino acid metabolism following liver injury: A study by means of paper chromatography. Quart. J. Med. n.s. 22: 483-505 (Oct.) 1953.
16. Dent, C. E.: Mechanisms of aminoaciduria. (Abstr.) Federation Proc., 6:390-391 (Mar.) 1947.
17. Salassa, R. M.; Power, M. H.; Ulrich, J. A., and Hayles, A. B.: Observations on the metabolic effects of vitamin D in Fanconi's syndrome. Proc. Staff Meet., Mayo Clin., 29:214-224 (Apr. 21) 1954.
18. Dent, C. E.: The amino-aciduria in Fanconi syndrome: A study making extensive use of techniques based on paper partition chromatography. Biochem. J., 41:240-253, 1947.
19. Sirota, J. H., and Hamerman, David: Renal function studies in an adult subject with the Fanconi syndrome. Am. J. Med., 16:138-152 (Jan.) 1954.
20. Lathem, Willoughby; Baker, Katharine, and Bradley, S. E.: Urinary amino acid excretion in renal disease, with observations on the Fanconi syndrome. Am. J. Med., 18:249-258 (Feb.) 1955.
21. Dent, C. E.; Heathcote, J. G., and Joron, G. E.: The pathogenesis of cystinuria. I. Chromatographic and microbiological studies of the metabolism of sulphur-containing amino-acids. J. Clin. Investigation, 33:1210-1215 (Sept.) 1954.
22. Dent, C. E., and Rose. G. A.: Aminoacid metabolism in cystinuria. Quart. J. Med., n.s. 20:205-219 (July) 1951.
23. Dern, P. L.: Amino-aciduria with cystinosis: Case report with determination of urinary amino acids and ocular cystine. Ann. Int. Med., 46:138-144 (Jan.) 1957.
24. Cooper, A. M.; Eckhardt, R. D.; Faloon, W. W., and Davidson, C. S.: Investigation of the aminoaciduria in Wilson's disease (hepatolenticular degeneration): Demonstration of a defect in renal function. J. Clin. Investigation, 29:265-278 (Mar.) 1950.
25. Uzman, L. L.: On the relationship of urinary copper excretion to the aminoaciduria in Wilson's disease (hepatolenticular degeneration). Am. J. M. Sc., 226:645-652 (Dec.) 1953.
26. Uzman, L. L.; Iber, F. L.; Chalmers, T. C., and Knowlton, Marjorie: The mechanism of copper deposition in the liver in hepatolenticular degeneration (Wilson's disease). Am. J. M. Sc., 231:511-518 (May) 1956.
27. Yeh, H. L.; Frankl, Willi; Dunn, M. S.; Parker, Paul; Hughes, Boland, and György, Paul: The urinary excretion of amino acids by a cystinuric subject. Am. J. M. Sc., 214:507-512 (Nov.) 1947.
28. Robson, Elizabeth B., and Rose, G. A.: The effect of intravenous lysine on the renal clearances of cystine, arginine and ornithine in normal subjects, in patients with cystinuria and Fanconi syndrome and in their relatives. Clin. Sc., 16:75-93 (Feb.) 1957.
29. Doolan, P. D.; Harper, H. A.; Hutchin, M. E., and Alpen, E. L.: Renal Clearance of Lysine in Cystinuria: Pathogenesis and management of this abnormality. Am. J. Med., 23:416-425 (Sept.) 1957.
30. Harris, H., and Robson, E. B.: Cystinuria. Am. J. Med., 22:774-783 (May) 1957.
31. Harrison, H. E., and Harrison, Helen C.: Aminoaciduria in relation to deficiency diseases and kidney function. J.A.M.A., 164:1571-1577 (Aug. 3) 1957.
32. Jonxis, J. H. P., and Huisman, T. H. J.: Amino aciduria and ascorbic acid deficiency. Pediatrics, 14:238-244 (Sept.) 1954.
33. Baron, D. N.; Dent, C. E.; Harris, H.; Hart, E. W., and Jepson, J. B.: Hereditary pellagra-like skin rash with temporary cerebellar ataxia, constant renal amino-aciduria, and other bizarre biochemical features. Lancet, 2:421-428 (Sept. 1) 1956.
34. Holzel, A.; Komrower, G. M., and Wilson, V. K.: Amino-aciduria in galactosaemia. Brit.M.J., 1:194-195 (Jan. 26) 1952.
35. Moore, S., and Stein, W. H.: Quoted by Jonxis, J. H. P., and Huisman, T. H. J.[32]
36. Dent, C. E.; Senior, B., and Walshe, J. M.: The pathogenesis of cystinuria. II. Polarographic studies of the metabolism of sulphur-containing amino-acids. J. Clin. Investigation, 33:1216-1226 (Sept.) 1954.
37. Weaver, J. A., and Neill, D. W.: Amino-aciduria in pernicious anaemia and subacute combined degeneration of the cord. Lancet, 1:1212-1213 (June 12) 1954.
38. Evered, D. F.: The excretion of amino acids by the human: A quantitative study with ion-exchange chromatography. Biochem. J., 62:416-427 (Mar.) 1956.
39. Dent, C. E.: Applications to study of amino-acid and protein metabolism. In: Biochemical Society Symposia No. 3: Partition Chromatography, pp. 34-50. Cambridge: University Press, 1949.
40. Brick, I. B.: The clinical significance of amino-aciduria. New England J. Med., 247:635-644 (Oct. 23) 1952.
41. Knox, W. E., and Hsia, D. Y.-Y.: Pathogenetic problems in phenylketonuria. Am.J.Med., 22:687-702 (May) 1957.
42. Allen, D. W., and Schroeder, W. A.: A comparison of the phenylalanine content of the hemoglobin of normal and phenylketonuric individuals: Determination by ion exchange chromatography. J. Clin. Investigation, 36:1343-1349 (Sept.) 1957.
43. Uzman, L. L., and Hood, B.: The familial nature of the amino-aciduria of Wilson's disease (hepatolenticular degeneration). Am.J.M.Sc., 223:392-400 (Apr.) 1952.
44. Iber, F. L.; Chalmers, T. C., and Uzman, L. L.: Studies of protein metabolism in hepatolenticular degeneration. Metabolism, 6:388-396 (July) 1957.
45. Berger, H., and Goldberg, A.: Hereditary coproporphyria. Brit. M.J., 2:85-88 (July 9) 1955.
46. Dent, C. E.: Foreword: to Symposium on inborn errors of metabolism. Am.J.Med., 22:671-675 (May) 1957.
47. Saint, E. G.; Curnow, D.; Paton, R., and Stokes, J. B.: Diagnosis of acute porphyria. Brit.M.J., 1:1182-1185 (May 22) 1954.
48. Rogers, H. M.; Keating, F. R., Jr.; Morlock, C. G., and Barker, N. W.: Primary hypertrophy and hyperplasia of the parathyroid glands associated with duodenal ulcer: Report of an additional case, with special reference to metabolic, gastrointestinal and vascular manifestations. Arch.Int.Med., 79:307-321 (Mar.) 1947.
49. Bell, G. O., and Arnold, W. T.: Primary hyperparathyroidism: Report of two unusual cases. Lahey Clin. Bull., 6:197-203 (Jan.) 1950.
50. Bogdonoff, M. D.; Woods, A. H.; White, J. E., and Engel, F. L.: Hyperparathyroidism. Am.J.Med., 21:583-595 (Oct.) 1956.
51. Cope, Oliver; Culver, P. J.; Mixter, C. G., Jr., and Nardi, G. L.: Pancreatitis, a diagnostic clue to hyperparathyroidism. Ann. Surg., 145:857-863 (June) 1957.
52. Plough, I. C., and Kyle, L. H.: Pancreatic insufficiency and hyperparathyroidism. Ann.Int.Med., 47:590-598 (Sept.) 1957.

Pathogenesis of Collagen Diseases

A General Review with Emphasis on the Face and Head

HENRY L. WILLIAMS, M.D.
Rochester, Minnesota

THE UPPER part of the respiratory tract extending from the lips and nasal vestibule to the larynx has not ordinarily been considered as a site of collagen disease. Semenov,[1,2] however, stated that certain disorders of the tunica propria of the nasal mucous membranes, particularly the type usually termed "hyperplastic sinusitis," should be included in the category of collagen diseases. He quoted Maximow and Bloom as saying that there are four main types of connective tissue, all characterized by abundant intercellular substance: (1) blood and lymph, (2) connective tissue proper, (3) cartilage and (4) bone.

In the connective tissue proper, the intercellular substance always contains fibers and varies from a soft jelly to a tough fibrous mass. Klemperer and associates[3] used the term "collagen system" to refer to the fiber-forming connective tissues throughout the body. Such tissue occurs in the nose and throat, particularly in the nasopharynx, the reticulum of tonsils and adenoids, and laryngeal polyps. One of the most obvious examples of collagen disease according to Semenov is nasal polyposis. He stated that polyps, whether bacterial or allergic in origin, have a poor blood supply and a tremendous amount of edema, which begins with abnormal capillary permeability. Perennial allergy or chronic infection or both produce profound effects in the collagen substance and perpetuate the edema. Semenov also said that polypoid sinusitis is characterized by a tremendous amount of chronic edema or a lesser degree of edema with thickening and degeneration of collagen tissue, hyalinization of the basement membrane and infiltration by eosinophils, lymphocytes and plasma cells.

Although allergy is a common cause of polypoid sinusitis, it is not the only cause. Infection may lead to similar changes. Histologically, edema and increase in collagen substance, hyperplasia of connective tissue, and infiltration of leukocytes, eosinophils, plasma cells and lymphocytes may be seen in various proportions in the mucous membrane in polypoid sinusitis. The actual hyperplasia becomes evident when the edema subsides. Even though the pathologic changes are divided into exudative, proliferative and degenerative stages, the main feature is the amount of permanent thickening.

Semenov[1,2] has presented convincing evidence that the collagen tissues of the nose and throat may be involved in disease processes and that these disease processes are frequently allergic. This is not exactly the same as classifying the disease processes as collagen diseases or as demonstrating that the collagen dieases are, as he implies, frequently allergic in origin.

To clarify these points it appears necessary (1) to consider what Klemperer had in mind when he first presented the term "collagen diseases," (2) to follow the development of the concept of collagen disease and (3) to determine what is meant by the term "allergy." It is helpful also to review briefly the available information on the ground substance of connective tissue, with its fibers and fibrils.

Definition of Collagen Disease

Klinge,[4,5] in 1929 and 1933, introduced the concept that a group of systemic diseases affects connective tissue. He focused attention on the conspicuous changes of the intercellular components of connective tissue, the fibrinoid alteration of collagenous tissue and the myxomatous swelling of the ground substance. Observation of similar changes in connective tissue in animals made hypersensitive to foreign proteins led Rössle[6] and Klinge[5] to conclude that the tissue damage in rheumatic fever and rheumatoid arthritis was

---

Read at the Sixth International Congress of Otolaryngology, Washington, D. C., May 5 to 10, 1957.

From the Section of Otolaryngology and Rhinology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

caused by hypersensitivity. Klinge stated that the same pathogenic mechanism was present in periarteritis nodosa, dermatomyositis, malignant nephrosclerosis, thromboangiitis obliterans, certain nephritides and cardiovascular sepsis.

Klemperer and co-workers[3] suggested the collective term "collagen diseases" to indicate the site of the characteristic lesion in these diseases, namely fibrinoid necrosis of the collagen fibers. Klemperer[7] later stated that when he and his associates originally proposed the term "collagen diseases" they were aware that the structural alterations which had been disclosed by conventional techniques of microscopic anatomy required further analysis. He considered that the term was not applicable to diagnosis and certainly did not apply to all the morbid processes of these diseases; all he wanted to express originally was that anatomic investigations in certain diseases disclosed conspicuous systematic changes in the intermediary substances of the connective tissue. Klemperer[8] also stated that a biologico-anatomic synthesis so sweeping as to include the collagen diseases among the allergic maladies was premature. He referred to the observations of Byron and Dodson, who produced fibrinoid necrosis in the arteries of normal animals by brief increases in intra-arterial tension. A number of other workers also have produced collagen diseases in animals by methods that probably excluded the factor of hypersensitivity.

Klemperer[7] regarded the connective tissue as the common seat of these heterogeneous maladies. He did not deny the probable role of hypersensitivity but was interested in a search for all factors that might be responsible for the conspicuous structural alterations; he also was interested in the mechanism of their action. It appeared obvious that such an inquiry had to take its origin from existing information concerning the structure and biology of normal connective tissue. He pointed out two problems that have yet to receive an unequivocal solution, namely the mode of fiber formation, and the nature and site of fabrication of the homogeneous ground substance.

Ground Substance and Fibers of Connective Tissue

Connective tissue is made up of an amorphous ground substance, mucinous in nature, containing collagen fibers. As Duran-Reynals[9] stated, the ground substance of the mesenchyme is an entity as old as histology itself, although it now may appear as a new one. The mesenchyme is likely to be considered an inferior structure that serves, depending on the circumstances, as a support to the passageway or as a lubricant. In the present concept, the ground substance is considered a part of other structures but as having also its own physiognomy and as functioning as a coherent unit.

The ground substance is a plastic material existing apparently in the form of a gel. In young undifferentiated connective tissue, ground substance is abundant and continuous; in adult subcutaneous tissue, it is less plentiful, being concentrated about the collagen fibers. The gel is greatly hydrated and therefore involved in water binding. Bensley[10] stated that the intercellular ground substance of loose connective tissue is a continuous, fairly granular, transparent material in which fibers are imbedded. It is elastic and tends to retract when cut. It is viscous and possibly acid. It is digested by trypsin but not by pepsin; the reverse is true of collagen. It combines with copper salts, suggesting that it may contain a derivative of chondroitin sulfuric acid.

Vaubel[11] found a synovioblast that apparently secreted mucin and was the source of the ground substance. He found also that secretion of mucin diminishes when synovioblasts change into fibroblasts. The mucinous nature of the homogeneous ground substance has been known for decades.

Kling[12] considered that the production of mucin was the result of cellular activity and that in inflammation the number of cytoplasmic granules in the synovioblasts of Vaubel and the secretion of mucin increase greatly.

Volterra[13] demonstrated that the adventitia is an indispensable element of all capillaries. Impregnation with silver shows that the adventitia consists of a delicate reticulum spread on and merged with an amorphous fundamental substance; the whole structure forms a complete membrane composed of what Volterra called "lamellated reticular tissue," which is continuous with the fibers of the surrounding connective tissue. Chambers and Zweifach[14] noted that the cement present between the endothelial cells of the capillaries is a sticky argyrophilic substance, continuously secreted by the cells themselves and

conditioned by calcium salts and the pH. They found that the cement behaves as a salt of calcium, that it is digested by trypsin and that it is not laid down in the absence of vitamin C.

Duran-Reynals[15] stated that the permeability of the connective tissue can be increased by certain factors and decreased by others. He found that the most important effect in increasing this permeability is that of the spreading factors, some of which are identified with mucolytic enzymes.

McMaster and Parsons[16] injected vital dyes into the connective tissues and noted that in the absence of edema the dye escapes from the lymph vessels as bristly, wavy lines of color formed by the dye moving between or along the fibrils of the connective tissue. The fluid moves through the tissues in thin films "captured" by capillary forces as if caught between two pieces of glass. As Duran-Reynals[15] pointed out, despite the viscosity of the ground substance, metabolites must move easily through it, impelled by capillary forces.

The investigations of Meyer[17] have greatly advanced knowledge of the mucopolysaccharides that enter into the constitution of the ground substance. They are mainly hyaluronic acid and chondroitin sulfuric acid. Meyer and Palmer[18] found a polysaccharide in the vitreous humor of the eyes of cattle consisting of hexosamine (d-glucosamine), hexuronic acid (glucuronic) and an acetyl. They named this product "hyaluronic acid." Meyer[17] suggested as a tentative feature of the development of cement substance in mesodermal tissues that the young fibroblast secretes hyaluronic acid, a precursor of collagen, and a chondroitin sulfate. By local acidification in the immediate neighborhood of the cells, the first fibers are produced by the polysaccharides from the native soluble collagen, which denatures into the soluble fiber, on the surface of which lies a sheet of polysaccharides. With aging, the polysaccharide layer becomes thinner and the hyaluronic acid is replaced more and more by chondroitin sulfate. Only in metabolically active tissues like that of skin does production of hyaluronic acid continue in appreciable quantities.

The fibers of connective tissue are divided into collagenous, elastic and reticular types. Studies with the electron microscope have shown that connective tissue contains many more fibers than was previously suspected. Even the smallest collagen fibers visible with ordinary microscopes are composed of bundles of minute fibrils, each of which exhibits periodic cross striations when viewed with the electron microscope.

Klemperer[8] pointed out that, while many experiments suggest that both the ground substance and the fibers of connective tissue are the result of cellular activity, the possibility that the ground substance is a transudate from the capillaries and that fibrin and collagen fibers can be formed from plasma clot by mere mechanical factors has not been completely ruled out.

Hass and McDonald[19] stated that an interaction between fibroblasts and ground substance resulted in formation of collagen. Robertson and Schwartz[20] confirmed the results of numerous histologic studies demonstrating the necessity of ascorbic acid for formation of collagen. They also found that the rapid formation of tissue containing large amounts of collagen increased the nutritional requirements of guinea pigs for ascorbic acid. The ascorbic acid in the process of fiber formation may be a component of chondroitin sulfate and may replace in the chain some of the molecules of glucuronic acid.

Ehrich[21] stated that the production and maintenance of connective tissue depend not only on the activity of fibroblasts and of the import of building stones from the blood, but also on regulating principles, such as enzymes, vitamins and hormones. The enzymes that degrade hyaluronic acid and chondroitin sulfuric acid are the hyaluronidases. They appear to be mixtures of several enzymes, some depolymerizing the long-chain molecules, others hydrolyzing aldobionic acid. The action of hyaluronidases *in vivo* is conditioned by the presence of antihyaluronidases. Ascorbic acid is outstanding among the vitamins that control connective tissue. Ehrich noted that thyrotropic hormone can stimulate the production of both ground substance and collagen fibers. However, he found that the action of adrenocorticoids had special effect.

Pathologic Physiology of Collagen Diseases

In discussing the pathology of connective tissue, Ehrich[21] stated that the alterations are degenerative or proliferative. The degenerative changes are mucoid degeneration, fibrinoid degeneration and necrosis, amyloidosis and paraamyloidosis. The proliferative change is the

secondary proliferation of connective-tissue cells, which is a common sequel of degeneration and necrosis of both connective tissue and granulation tissue.

Primary proliferation of connective-tissue cells occurs in myxedema and generalized scleroderma and probably in other collagen diseases. Ehrich found severe primary proliferation to be a characteristic feature of subacute allergic reactions (granulomas). Allergic proliferation is usually located in the intima or adventitia of blood vessels.

Ehrich also reviewed the pathologic physiology and morphology of each collagen disease, including rheumatic fever, rheumatoid arthritis, lupus erythematosus disseminatus, generalized scleroderma, dermatomyositis, serum sickness and periarteritis nodosa. He considered that rheumatic fever probably is an allergic response to either group A hemolytic streptococci or their products. He stated that the cause was unknown in rheumatoid arthritis, lupus erythematosus, generalized scleroderma and dermatomyositis. He remarked that it is well established that serum sickness is an allergic reaction due to intravenous injection of foreign serum or other foreign proteins and that the arterial changes in periarteritis nodosa resemble those in serum sickness so closely that the two appear to be the same; thus, he concluded that periarteritis nodosa is an allergic disease due to an Arthus reaction.

Long[22] stated that the collagen diseases are systemic, not local, conditions. Klinge[4] considered fibrinoid necrosis to be presumptive evidence of a hypersensitivity reaction. He thought that systemic diseases exhibiting fibrinoid necrosis have not been shown to occur in the absence of naturally occurring or artificially induced allergic reactions. The observation that fibrinoid necrosis may develop locally as a result of mechanisms other than allergy did not in Long's opinion disprove Klinge's hypothesis. Long stated that allergic reactions are of two types, namely immediate (anaphylactic), which is either systemic or local, and delayed, which is the result of bacterial allergy (tuberculin type); in his opinion, the delayed type of hypersensitivity reaction is the one present in collagen diseases. He stated that it is a normal sequence of any bacterial infection, so that it is not necessary to postulate an abnormal hypersensitivity reaction. It is a natural phenomenon in which sensitivity is induced by persistent low-grade infection. The response is delayed and is due to antibody fixed on the cells rather than to circulating antibody, as is the case in the immediate type of reaction.

Rich,[23] in discussing hypersensitivity in disease, stated that there is high promise in an attack on the virtually untouched fundamental problems relating to the intimate chemical mechanisms through which hypersensitive reactions exert their injurious effects on the tissues.

In a review of the possible allergic mechanisms involved in collagen diseases, Miale[24] stated that necrosing arteritis in man has been preceded or accompanied by bronchial asthma, a variety of allergic states, acute and chronic bacterial infections, protozoan infestation, administration of drugs and serum sickness. Since in only one or two of these situations is it possible to show qualitative and quantitative similarities to the Arthus experiment, it follows that vascular necrosis does not always represent an Arthus reaction but is more often an expression of other types of allergic reactions.

Rössle,[6] in observing by biomicroscopy the changes of "allergic inflammation" in the mesentery of the frog, noticed that the first reaction was in the blood flow. He found degenerative changes in the walls of small blood vessels, with deposits of fibrin in zones of edema. Following these changes, the fibrin was replaced by proliferative fibrocapillary tissue. Harkavy,[25] in pointing out the importance of the vascular reactions in the symptomatology of allergy, stated that when dealing with bacterial allergy the characteristic symptoms of the disease to which the patient is subject may be reactivated by contact with homologous bacteria responsible for the initial sensitizing effect, and also possibly by viruses and by heterologous bacteria and their toxins, as well as by antigen-antibody complexes of nonbacterial origin.

Several years ago, I[26] suggested that, because the allergic reaction made use of a stereotyped (invariable) vascular mechanism in producing its results and because an antigen-antibody component has not been demonstrated in the lesions of physical allergy, the immunologic mechanism might be supposed to produce so-called allergic responses without an antigen-antibody reaction being present. I thought that this might explain

the infrequency of favorable clinical results from attempts at hyposensitization.

From a review of the literature, it is apparent that knowledge of the mechanisms of conditions considered to be allergic is still in its infancy. The difficulty that we physicians face when we treat patients with allergy is that we are trying to apply to clinical medicine a concept that is still in the stage of investigation. The concept of physical allergy presented by Weed never has been successfully controverted. Interesting hypotheses have been presented recently in which the altered reactivity in "allergy" is considered as not due to an antigen-antibody reaction but rather to an enzymatic disorder.

Jaros,[27] in presenting a hypothesis on the physiochemical pathogenesis of the collagen diseases, stated that availability of purified adrenocorticoids has unfolded a new era in medicine. In evaluating the pharmacologic effectiveness of such compounds, he considered that it was immediately apparent that epinephrine, acetylcholine and histamine are involved. The relationship of these three substances to the enzymes and to adrenocorticoids formed the basis for a new hypothesis. He emphasized that the abnormal mechanism involved apparently was present only in persons who had inherited a predisposition to collagen diseases. It was his belief that the tissues in such persons have an abnormal responsiveness, which is exhibited by exaggerated reactions toward acetylcholine and histamine formed in the sensitized end-organs. He suggested that the pathologic reaction and the attempt at adaptation occur in two phases. The catabolic phase concerns the deleterious effects produced in the tissues and is due to the incompletely antagonized acetylcholine and histamine, whereas the anabolic phase involves the adrenocorticoids, which enhance the formation of enzymes that inhibit or neutralize the excess of acetylcholine and histamine present. He suggested that the best management of the collagen diseases should include the use of cholinesterase or other efficient antiacetylcholine and antihistaminic drugs.

Godlowski[28] suggested that the manifestations of anaphylaxis (allergy) are not directly caused by the antigen-antibody interaction. He suggested that in the enzymatic concept of anaphylaxis the anaphylactic phenomena should be regarded rather as resulting from the failure of the antigen-antibody reaction.

Collagen Diseases of the Face and Head

From this review of the literature, it appears that hyperplastic sinusitis cannot be classified as on of the collagen diseases described by Klinge and by Klemperer. Although the proliferative and degenerative changes in the tunica propria seen in the collagen diseases are present in hyperplastic sinusitis, it hardly appears warranted to consider it as a generalized disorder, even though, according to Semenov, it often may be due to the bacterial type of allergy.

However, two disorders occur in the region of the head, one in the upper part of the respiratory tract and the other involving the eye and inner ear, that meet the demands of strict classification as collagen diseases. These are lethal granulomas of the midline facial tissues, and nonsyphilitic interstitial keratitis and deafness (Cogan's disease). In these conditions, the primary lesion is associated with generalized periarteritis nodosa.

Krompecher[29] reviewed the literature on destructive midline lesions of the face. His description of the pathologic changes is one that might correspond to what is now known as syncytial cell sarcoma. Kraus,[30] in 1929, described three cases of a "special neoformation" of the nasal cavities and suggested that destructive midline facial lesions might be granulomatous rather than malignant. MacNaughton-Jones in 1897,[31] apparently was the first to describe a granulomatous lesion of the midline facial tissues. In 1931, Wood[32] presented the first case of malignant granuloma reported in the North American literature. Wegener,[33] in 1939, described the association of midline granuloma with periarteritis nodosa, an association that since has been confirmed on numerous occasions.

Terracol and Camps,[34] in a scholarly and critical review of the literature, analyzed reports of seventy-four cases. They assumed that twenty-four of these lesions were due to an infectious process and twenty-one were malignant tumors, of which fourteen were reticulum cell sarcomas and seven were lymphosarcomas; six lesions were "malignant reticulosis" and sixteen were caused by an allergic affection. They considered that malignant granuloma ought not to be considered

as an idiopathic disorder but as a syndrome of multiple causes.

Di Pietro[35] reported a case of nasal granuloma from which Rhinosporidium seeberi was isolated. De Faria and co-workers[36] reported three lesions resembling malignant granuloma that showed a histologic picture of reticulosarcoma. They suggested that all cases of "malignant granuloma" were of this type. However, none of the lesions for which a definite cause could be proved were associated with generalized periarteritis nodosa.

Midline ulcerations not associated with the generalized lesions of periarteritis nodosa would appear usually to be of a different cause than the true malignant granuloma with generalized periarteritis nodosa, although the local clinical appearance may be the same.

Because of the association of certain of these midline facial lesions with periarteritis nodosa, it was suggested by Williams and Hochfilzer[37] that administration of anti-inflammatory corticoids might reverse the process. Moore and associates[38] reported a case in which use of cortisone completely cleared up the granulomatous lesions in the nose, palate and pharynx. Although the granulomatous lesions did not recur, the patient died a year later from cardiovascular renal disease considered to be the result of healing of the lesions of the associated periarteritis nodosa.

Hagens and co-workers[39] reported a case in which malignant granuloma of the nose and face was treated with corticotropin (ACTH) and has remained healed for four years.

Vaheri[40] reported a malignant granuloma in a sixteen-year-old girl. Numerous eosinophils were found on biopsy of the lesion, and the patient had a blood eosinophilia of 10 per cent. The lesions involved the left orbit, left posterior pillar and left tonsil. Rapid improvement with use of cortisone was noted.

Summary

Review of the literature indicates that the reactions of connective tissue, especially the ground substance of the mesenchyme and the collagen fibers, are of great importance in certain diseases of a generalized type in which proliferative and degenerative changes occur in these tissues, especially in the walls of the blood vessels.

These reactions are stereotyped and invariable and suggest a common mechanism, which may be anaphylactic or allergic, particularly of the bacterial type. However, these reactions can be found when an antigen-antibody reaction cannot be demonstrated. Therefore, if these conditions are to be thought of as allergic, allergy must be considered in its broadest sense as an "unusual reaction" and not strictly as an antigen-antibody reaction.

The only conditions in the region of the head that strictly fit the definition of collagen disease are lethal granuloma of the midline facial tissues, and the syndrome of nonsyphilitic interstitial keratitis and deafness. However, polypoid hyperplasia with collagenous degeneration of the tunica propria of the nasal mucosa, although not associated with a generalized reaction, is a similar type of reaction and is known to respond favorably to cortisone and corticotropin. It is usually an allergic reaction of connective tissue, although it is not a "collagen disease."

References

1. Semenov, Herman: Pathology of nose and paranasal sinuses in relation to allergy with comments on local injection of cortisone. Tr. Am. Acad. Ophth., 56:121-170 (Mar.-Apr.) 1952.
2. Semenov, Herman: The pathology of the nose and paranasal sinuses. II. Rhinitis and sinusitis. Tr. Am. Acad. Ophth., 57:399-440 (May-June) 1953.
3. Klemperer, Paul, Pollack, A. D., and Baehr, George: Diffuse collagen disease. Acute disseminated lupus erythematosus and diffuse scleroderma. J.A.M.A., 119:331-332 (May 22) 1942.
4. Klinge, Fritz: Die Eiweifsüberempfindlichbeit (Gewebs-anaphylaxie) der Gelinke. Experimentelle pathologisch-anatomische Studie zur Pathogenese des Gelenkrheumatismus. Beitr. z. path. Anat., 83: 185-216 (June) 1929.
5. Klinge, Fritz: Der Rheumatismus. Pathologisch-anatomische und experimentelle-pathologische Tatsachen und ihre Auswortung für das ärztliche Rheumaproblem. Ergebn. d. allg. Path. u. path. Anat., 27:1-351, 1933
6. Rössle, R.: Originalabhandlungen. Die geweblichen Äusserungen der Allergie. Wien. klin. Wchnschr., 45:609-612 (May 13); 648-651 (May 20) 1932.
7. Klemperer, Paul: The concept of collagen diseases. Am. J. Path., 26:505-519 (July) 1950.
8. Klemperer, Paul: Diseases of the collagen system. Bull. New York Acad. Med., 23:581-588 (Oct.) 1947.
9. Conference on: The ground substance of the mesenchyme and hyaluronidase. Chairman: Duran-Reynals, F. Ann. New York Acad. Sc., 52:943-1196 (May 31) 1950.
10. Bensley, Sylvia H.: On the presence, properties and distribution of the intercellular ground substance of loose connective tissue. Anat. Rec., 60:93-108 (Aug.) 1934.
11. Vaubel, Ernest: The form and function of synovial cells in tissue cultures. II. The production of mucin. J. Exper. Med., 58:85-95 (July) 1933.
12. Kling, D. H.: The Synovial Membrane and the Synovial Fluid With Special Reference to Arthritis and Injuries of the Joints. Los Angeles: The Medical Press, 1938.
13. Volterra, Mario: Einige neue Befunde über die Struktur der Kapillaren und ihre Beziehungen zur "Sogenannten" Kontraktiletät derselben. Zentralbl. f. inn. Med., 46:876-881 (Aug.) 1925.

14. Chambers, Robert and Zweifach, B. W.: Capillary endothelial cement in relation to permeability. J. Cell. & Comp. Physiol., 15:255-272 (June) 1940.
15. Duran-Reynals, F.: Tissue permeability and spreading factors in infection; a contribution to the host: Parasite problem. Bact. Rev., 6:197-252 (Dec.) 1942.
16. McMaster, P. D., and Parsons, R. J.: Physiological conditions exisfing in connective tissue. I. The method of interstitial spread of vital dyes II. The state of the fluid in the intradermal tissue. J. Exper. Med., 69:247-282 (Feb.) 1939.
17. Meyer, K.: Biological significance of hyaluronic acid and hyaluronidase. Physiol. Rev., 27:335-359 (July) 1947.
18. Meyer, Karl and Palmer, J. W.: The polysaccharide of the vitreous humor. J. Biol. Chem., 107:629-634 (Dec.) 1934.
19. Hass, George, and McDonald, Francis: Studies of collagen I. The production of collagen *in vitro* under variable experimental conditions. Am. J. Path., 16:525-548 (Sept.) 1940.
20. Robertson, W. V., and Schwartz, Barry: Ascorbic acid and the formation of collagen. J. Biol. Chem., 201:689-696 (Apr.) 1953.
21. Ehrich, W. E.: Nature of collagen diseases. Am. Heart J., 43:121-156 (Jan.) 1952.
22. Long, D. A.: Discussion on Hypersensitivity and the collagen diseases. Proc. Roy. Soc. Med., 49:295-297 (May) 1956.
23. Rich, A R.: Hypersensitivity in disease, with especial reference to periarteritis nodosa, rheumatic fever, disseminated lupus erythrematosus and rheumatoid arthritis. Harvey Lect., 42:106-147, 1946-1947.
24. Miale, J. B.: The manifestations and mechanisms of vascular allergy: A critical review. Ann. Allergy, 7:124-149 (Jan.-Feb.) 1949.
25. Harkavy, Joseph: Vascular allergy. III. J. Allergy, 14:507-537 (Nov.) 1943.
26. Williams, H. L.: A concept of allergy as autonomic dysfunction suggested as an improved working hypothesis. Tr. Am. Acad. Ophth., 55:123-146 (Nov.-Dec.) 1951.
27. Jaros, S. H.: A hypothesis on the physiochemical pathogenesis of hypersensitivity states and collagenous diseases. Ann. Allergy, 9:113-150 (Mar.-Apr.) 1951.
28. Godlowski, Z. Z.: Enzymatic Concept of Anaphylaxis and Allergy and the Role of Eosinophils in Anaphylactic Reactions Related to Hormonal Alterations. Baltimore: Williams & Wilkins Company, 1953.
29. Krompecher, E.: Der drüsenartige Oberflächenepithelkrebs. Carcinoma epitheliale adenoides. Beitr. path. Anat., 28:1-41, 1900.
30. Kraus, E. J.: Über ein eigenartiges granulom der Nasen-, Racken- und Mundhöhle. Verhandl. d. deutsch. path. Gesellsch., 24:43-57, 1929.
31. MacNaughton-Jones: Esthiomenic menstrual ulcer of nose. J. Laryng. & Otol., 12:301-306 (June) 1897.
32. Wood, G. B.: A case of mutilating granuloma of the nose and face with fatal ending. Tr. Am. Laryng. A., 53:63-71, 1931.
33. Wegener, F.: Über eine eigenartige rhinogene Granulomatose mit besonderer Beteilegung des Arteriensystems und der Nieren. Beitr. path. Anat., 102:36-68, 1939.
34. Terracol, J., and Camps, F.: Le mésenchymome malin des tissus médio-faciaux. Montpellier méd., 45:75-175 (Feb.) 1954.
35. di Pietro, Arturo: Granuloma nasal a Rhinosporidium seeberi (8ª observacion argentina). Arch. Soc. argent. de anat. norm. y. pat., 6:169-178 (June 16) 1944.
36. de Faria, J. L., Cutin, Moyses, and Morgante, Paulo: Malignant granuloma of the face: Contribution to its nosology. A.M.A. Arch. Otolaryng., 65:255-262 (Mar.) 1957.
37. Williams, H. L., and Hochfilzer, J. J.: Effect of cortisone on idiopathic granuloma of midline tissues of the face. Ann. Otol. Rhin. & Laryng., 59:518-530 (June) 1950.
38. Moore, P. M., Beard, E. E., Thoburn, T. W., and Williams, H. L.: Idiopathic (lethal) granuloma of the midline facial tissues treated with cortisone: Report of a case. Laryngoscope, 61:320-331 (Apr.) 1951.
39. Hagens, E. W., Parry, N., and Markson, D.: Corticotropin (ACTH) in lethal granuloma of nose and face. A.M.A. Arch. Otolaryng., 57:516-519 (May) 1953.
40. Vaheri, E.: Cortisone treatment in malignant granuloma. Pract. oto-rhino-laryng., 18:49-59 (Jan) 1956.

DETECTION OF POLYPS, POSSIBLE FORERUNNERS TO CANCER

By the early detection of small growths, called "polyps," in the large intestine, radiologists can help reduce deaths from intestinal cancer. The polyps are apparently often the forerunners of cancer. Once detected they can be removed surgically.

This was the comment of Dr. J. Maurice Robinson, San Francisco radiologist, in the April, 1957 issue of the *American Journal of Roentgenology, Radium Therapy and Nuclear Medicine.*

Dr. Robinson described x-ray procedures for these sometime obscure growths. He believes the procedure helps detect them more effectively.

"It has seemed evident that the problem of reducing the mortality of cancer of the large bowel and rectum, now responsible for 17 per cent of all deaths from cancer, is closely linked to the detection and eradication of the polyp, which is apparently the precursor of the malignant," Dr. Robinson explained.

After describing in detail the technical aspects of x-ray diagnostic procedures, Dr. Robinson added this warning: "The author has been forced" to the unhappy conclusion that there are unavoidable pitfalls inherent in any of these methods. The procedure which works well on one day will not work well on another; and on some days, none of them seems to be any good."

Other conclusions:

—One important element in assuring moderate success in finding these tricky growths is the earnest conviction on the part of the diagnostic radiologist that it is *important* to find them.

—Concentration of attention should be on the "happy hunting ground" which is the remote third of the large bowel. This is where 70 per cent of the polyps are located, in patients over fifty years old.

Even before the polyps have produced such physical symptoms as bleeding, radiologists have been able to locate them, previous reports indicate. "They appear to be punched out, as though with a cookie-cutter, and they seem to stand out much more sharply than one would expect. . . . Not infrequently the polyp defect shows as a very dense black center, the remainder of the defect being somewhat less dense."

Cooperation of Community Blood Banks in Supplying Blood for a Sensitized Hemophiliac Patient

G. A. MATSON, Ph.D.
L. J. HAY, M.D.
R. J. WEISBERG, M.D.
Minneapolis, Minnesota

THE ROLE OF community blood banks in supplying blood for the everyday needs of patients in hospitals they serve, is well understood. Little or no difficulty is experienced ordinarily in providing an adequate supply of fresh blood or blood derivatives for the average patient. A real problem in blood procurement may present itself, however, if a patient having an unusual blood-group formula requires blood in large amounts. At such a time there is a great advantage in being able to call upon other blood banks for assistance in the specific problem encountered rather than to rely on an appeal to the public by radio, television or the press, which at best yields only an assortment of unselected bloods. The present report describes the satisfactory management of a problem that occurred at the Minneapolis War Memorial Blood Bank in supplying blood and fresh frozen plasma for a sensitized hemophiliac patient with a bleeding ulcer. The case is cited as an example of what can be accomplished when several community blood banks work together.

Case History

A thirty-nine-year-old man was admitted to a Minneapolis hospital on December 8, 1955, because of bleeding from a gastric ulcer. During the prior eighteen years, the patient had been hospitalized many times for various illnesses and injuries, which included trauma to the knees and other joints, dental surgery, appendectomy and five episodes of bleeding from the gastric ulcer. He had received between thirty-five and forty transfusions, several of which had caused reactions. During these many hospitalizations, a diagnosis of hemophilia had been made.

During the current admission, studies of the patient's plasma showed a prolonged plasma coagulation time, which was greatly shortened by the addition of fresh normal plasma and of plasma treated with barium sulfate, but which was not influenced by the addition of fresh serum.[1] Attempts at crossmatching at the hospital indicated the presence of abnormal blood-group antibodies. The patient's blood was sent to the Minneapolis War Memorial Blood Bank for cross-matching and studies.

Serological Findings.—The blood-group formula of the patient for the factors tested was group 0, Rh-positive (probable genotype R^1R^1 [CDe/CDe]), P-, K-, Fy(a-). It was apparent that he lacked rh″(E), hr′(c), P, K and Fy(a+), to any one or more of which factors he could have been sensitized. The significant reactions of patient and donor test cells to antisera and of the patient's serum to these cells are shown in Figure 1.

The results in this table showed that the patient had multiple antibodies: anti-hr′(anti-c), anti-rh″(anti-E) and anti-Duffy(anti-Fy^a). Antibody titers were determined on the patient's serum. It contained anti-hr′(anti-c) in a titer of 2 in saline, 32 in albumin and 64 by the indirect Coombs' test. The anti-rh″(anti-E) was demonstrable in albumin in a titer of 1 and by the indirect Coombs' test in a titer of 4. The anti-Duffy (anti-Fy^a) titer was 2 in saline, 1 in albumin and 4 by the indirect Coombs' test. A probable dosage effect was observed in the early specimens. Duffy cells were agglutinated in saline, whereas others were demonstrable only by the indirect Coombs' test; the latter were probably heterozygous. Evaluation of the requirement of antihemophilic globulin indicated that clotting times were normal at intervals of one, five and eleven hours after administration of 100 cc. of fresh frozen plasma.

Medical Management

The medical management of this present episode of gastric bleeding was successfully accomplished without complications and without transfusion reactions. At this point, a decision relative to definitive surgical treatment had to be made. The situation presented a unique problem in evaluating relative risks. This patient, a hemophiliac now thirty-nine years old with a history of five previous episodes of gastric bleeding, faced a progressively greater risk of future medical or surgical failure as his hemophilia became complicated by arteriosclerosis. If his future management was dependent on blood transfusions and fresh plasma, the possibility existed that he would not only acquire new sensitivities to other blood antigens but also acquire antibodies against the antihemophilic globu-

From the Minneapolis War Memorial Blood Bank and Mt. Sinai Hospital, Minneapolis, Minnesota.

lin itself. Finally, because of the already existing multiple sensitizations, there was the great problem of supplying the specific blood type in amounts adequate to control a severe acute episode of bleeding wherever, geographically, the next emergency occurred. Since about 17 per cent of Western Europeans are homozygous rh′ (C) and homozygous hr″ (e), and about 30 per cent are Duffy (Fy^a) negative, one could expect to find about 5 per cent of group O bloods to be compatible with the patient. In Minnesota, about 40 per cent of bloods belong to group O; therefore, about 2 per cent of blood donors selected at random would have the righ blood formula to be compatible with that of the patient.

After weighing these risks against the well-known dangers of doing a major surgical operation upon a hemophiliac patient, it was the opinion that elective surgical intervention in the interval between bleeding episodes was the lesser risk. Accordingly, gastric resection was done on December 26. Six units of compatible whole blood were found in the bank's entire inventory and these, together with large amounts of fresh frozen plasma, were on hand at the time of operation. Hemostasis at operation was meticulous. The smaller bleeding points were controlled by coagulation and the larger ones by ligation. Nevertheless, the incision, though apparently dry when closed, soon began to bleed continuously. On the second postoperative day, a very small cutaneous bleeder was ligated after spreading the incision for 2.5 centimeters. Gastrointestinal bleeding was evident from the ninth to the fourteenth postoperative day and again on the twenty-third day. On the twenty-sixth day after operation, disruption of the middle third of the wound occurred. Little evidence of healing was present, the wound having the appearance of one separating on the seventh or eighth day. No evidence of infection or wound hematoma was noted. The wound surfaces appeared healthy, but little fibroplasia was evident. The disruption was sutured, but the healing continued to be slow. The retention-suture holes bled frequently, and the wound was not healed firmly until after a period of five months.

SIGNIFICANT REACTIONS* OF CELLS AND PATIENT'S SERUM

Anti-sera											Patient's serum				
											Crossmatches				
												Saline	Albumin		
Cells	Blood Group	Rh_o D	rh′ C	rh″ E	hr′ c	hr″ e	P	K	Fy^a	Papain Screening	37°	20°	37°	4°	I.C.T.
Patient L.S.	O	+	+	−	−	+	−	−	−		−	−	−	+ −	−
R.H.	O	+	−	−	+	+	+	−	+	4+	+	+	4+		4+
R.S.	O	−	+	−	+	+	+	−	−	4+	+	+	4+		4+
R.K.	O	−	−	+	+	+	+	+	+	4+	+	+	4+		4+
F.H.	O	−	−	−	+	+	−	−	−	4+	+	+	4+		4+
8196	O	+	+	−	+	+	−	−	−		+	+	4+		4+
26470	O	+	+	−	−	+	+	−	+		+	2+	+		3+
8440	O	+	+	−	−	+	+	+	+		−	−	−		3+
8399	O	+	+	−	−	+	+	+	−		−	−	−		−

*Reactions of cells to antisera M, N, S, s, Le^a, Le^b, k, Fy^b, Lu^a, and Jk^a are known for some of the bloods on the table, but these are not significant and therefore not shown.

Fig. 1. Reactions in a hemophiliac patient undergoing gastrectomy. I.C.T. indicates indirect Coombs' test.

Administration of Blood and Fresh Frozen Plasma.—Fresh frozen plasma and whole blood were given for eight weeks and the patient was hospitalized for nine weeks. During this time, the patient received plasma in increasing amounts

TABLE I. BLOOD AND PLASMA ADMINISTERED TO A HEMOPHILIAC PATIENT

Date	Postop. Day	Plasma cc.	Blood cc.	Comment
12-24		100		
12-25		200		
12-26		150	1900	Gastrectomy
12-27	1	585	1050	
12-28	2	400	1800	Wound bleeder caught
12-29	3	500	500	
12-30	4	500	500	
12-31	5	500		
1- 1	6	600		
1- 2	7	500	2000	
1- 3	8	900	500	
1- 4	9	1200	1450	
1- 5	10	1200	2900	
1- 6	11	1200	1500	G. I. Bleeding
1- 7	12	1100	1500	
1- 8	13	1200		
1- 9	14	1200	500	
1-10	15	1200		
1-11	16	1200		
1-12	17	1200		
1-13	18	1200		
1-14	19	1150		
1-15	20	650		
1-16	21			
1-17	22			
1-18	23	200		G. I. Bleeding
1-19	24	400	1000	
1-20	25	800	1500	
1-21	26	1200	2500	Wound separation
1-22	27	1500	3000	
1-23	28	1800		
1-24	29	1425		
1-25	30	1250		
1-26	31	1500		
1-27	32	1000		
1-28	33	1000		
1-29	34	900		
1-30	35	700		
1-31	36	1500		
2- 1	37	1200		
2- 2	38	1250		
2- 3	39	1150		
2- 4	40	850		
2- 5	41	1000		
2- 6	42	1000		
2- 7	43	800		
2- 8	44	900		
2- 9	45	700		
2-10	46	750		
2-11	47	1050		
2-12	48	950		
2-13	49	900		
2-14	50	900		
2-15	51	700		
2-16	52	600		
2-17	53	600		
2-18	54	400		
2-19	55	290		
2-20	56	300		
2-21	57	100		

from the day of operation, averaging 500 cc. per day until the eighth day, when 900 cc. was given. From the tenth through the nineteenth day, 1200 cc. per day was given. After four days at this level, the bleeding stopped. The amount of plasma was decreased to 650 cc. on the twentieth day, and its use was omitted on the twenty-first and twenty-second days. When bleeding from the gastrointestinal tract began again on the twenty-third day, fresh frozen plasma and blood again were administered. At the time of the wound separation on the twenty-sixth day, the volume of fresh frozen plasma was increased to more than 1200 cc. per day for the first six days after its repair. When bleeding was controlled, the intake was decreased to between 750 and 1000 cc. per day. The maximal amount of fresh frozen plasma given was 1800 cc. per day. The record of fresh frozen plasma and blood administered is shown in Table I and Figure 2.

Review of Table I and Figure 2 indicates that 1200 cc. of fresh frozen plasma per day was required to control bleeding in this patient. The clotting time was kept within normal limits when this amount was given. This agrees with the experience of Aggeler,[2] who pointed out that a 60-kg. person with a blood volume of 5000 cc. would require approximately 1500 cc. of fresh plasma given at the rate of about 300 cc. every two hours for the first ten hours to achieve a 30 per cent level of antihemophilic factor. After this initial dose, Aggeler recommended that approximately 1 cc. per kilogram of body weight per hour be given until all evidence of bleeding had disappeared.

Supplying Blood and Plasma

It became apparent soon after the operation had been completed that the patient would require large amounts of blood to replace the loss due to continuous seepage, as well as large amounts of fresh frozen plasma to overcome the deficiency of antihemophilic globulin.

More blood than the six units on hand surely would be needed and most likely in large amounts. An appeal was made to the Milwaukee Blood Center for group O, Rh_1, homozygous rh′ (C) and Fy(a-) bloods. During the illness of this patient, the Milwaukee bank shipped blood to Minneapolis on five different occasions, sending a total of thirty-seven units. These were crossmatched at the Minneapolis War Memorial Blood Bank and twenty-eight of them were used for the patient. In the meantime, the laboratory staff screened a total of 923 group O, Rh positive bloods, of which sixty proved to be compatible. Two units of blood were received from the Mt. Sinai Research Center, Chicago, and one from the John Elliot Blood Bank, in Miami. Fresh frozen plasma was prepared daily by the Minneapolis blood bank and administered at the hospital. During the seventy-one days of hospitalization, the patient used fifty-three units of whole blood, together with 203 units (50 cc.) and 423 units (100 cc.) of fresh frozen plasma. Converted to 500 cc. units of whole blood, the equivalent of 332 units was used.

The patient is now clinically well, having been released from the hospital on February 25, 1956.

This experience emphasized the inadequacy of any one blood bank to meet certain emergencies. The promptness and dispatch with which other community blood banks came to the aid of the Minneapolis War Memorial Blood Bank in this emergency demonstrated how such emergencies can be met and lives saved by co-operative efforts of good community blood banks. The credits for such transactions can be easily cleared by working through the American Association of Blood Banks' clearing houses.

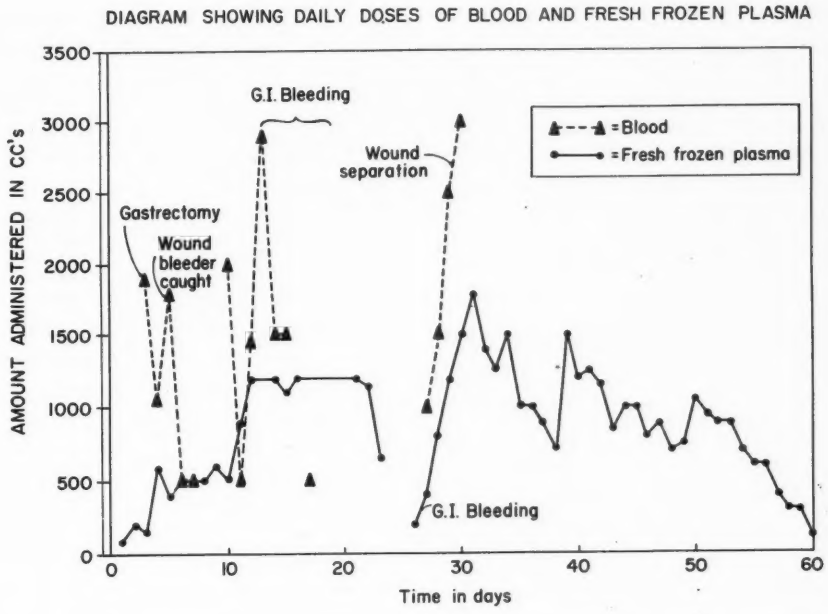


Fig. 2. Administration of plasma and blood to a hemophiliac patient undergoing gastrectomy.

Summary and Conclusion

The role of a number of community blood banks working together in meeting the blood needs in a case of elective subtotal gastric resection, in a hemophiliac patient who was sensitized against rh″(E), hr′(c) and Fy(a+), is described. The importance of community blood banks co-operating in emergencies is emphasized. Working through the American Association of Blood Banks' clearing houses simplifies such transactions.

Acknowledgment

Appreciation is expressed to Dr. Tibor Greenwalt, Milwaukee Blood Center, Inc., who confirmed our antibody findings and sent selected blood to be crossmatched for the patient. Gratitude is also expressed to Dr. Kurt Stern, Mount Sinai Research Center, Chicago, and to Dr. Lloyd Newhauser, John Elliot Blood Bank, Miami, for sending selected blood for this patient.

References

1. Owen, C. A., Jr., Mann, F. D., Hurn, Margaret M., and Stickney, J. M.: Evaluation of disorders of blood coagulation in the clinical laboratory. Am. J. Clin. Path., 25:1417-1426, 1955.
2. Aggeler, P. M.: Panels in therapy. IX. The treatment of hemophilia. Blood, 11:81-84, 1956.

Surgical Treatment of Esophageal Hiatus Hernia

A Description of Some Important Details in the Technique of Repair

FRANK E. JOHNSON, M.D.
Minneapolis, Minnesota

THE PROBLEM of hiatus hernia has been appreciated best in recent times. In fact, up until 1921 only forty-four cases of all types of diaphragmatic hernia had been diagnosed during life.[1] In 1926, Akerlund[2] collected sixty cases of hiatus hernia diagnosed during life and reported twenty-four of his own. This reflects the more general use of x-ray in diagnosis. Surgical treatment of hiatus hernia essentially dates from Harrington's report of his repair in sixty cases in 1933.[3] A significant advance in the technique for repair of this hernia was made when a more anatomical closure of the defect posterior to the esophagus was performed.[4,5]

Simple repair of the hernia is sufficient in the majority of cases, but does not always prevent or correct one of the important accompaniments of hiatus hernia, namely, reflux esophagitis with its attendant serious complications. This is apparent from the fact that rather extensive extirpative and reconstructive procedures have been advocated and performed in an effort to correct the complications of reflux esophagitis.[6,7] It is important to note that the changes consequent to esophageal reflux, even though severe, are reversible if the irritant is removed.[8] In the normal person, prevention of reflux has been attributed to actual compression of the esophagus by the right crural fibers as they course around the esophageal hiatus. This arrangement is the so-called diaphragmatic pinchcock[9] which could only function during those times that the diaphragm contracts. At operation, with a finger in the hiatus, a pinchcock effect is difficult to detect. Some observers have described a lower esophageal circular sphincter muscle.[10] This cannot be a constant or a definite structure since other competent observers have been unable to find it in their dissections.[11]

There is interesting experimental evidence that the angle of insertion of the esophagus to the stomach is the important factor in preventing reflux into the esophagus (Fig. 1).[12] The treatment of reflux esophagitis, medical as well as surgical, will have its greatest measure of success if it is based on this experimental evidence.

The indications for surgery in the treatment of hiatus hernia are similar to those for duodenal ulcer, namely, symptoms intractable to medical management or complications of a serious nature such as gastrointestinal bleeding (chronic or acute), esophageal stenosis, gastric ulcer at the neck of the hernia, or perforation of the stomach or esophagus. In some of the author's cases, indication for operation was based on the objective finding of pathology present in other organs, most often the gall bladder.

The symptoms in these cases, however, were characteristic of reflux esophagitis. In weighing the case for surgical treatment in this disorder one notes: (1) that the problem is a mechanical one and might reasonably be expected to respond incompletely to medical therapy, (2) that hiatus hernia increases in size with time, and (3) that Blades[13] reports 45 per cent of his series of sixty-six cases had dangerous complications (bleeding, twenty-two; stricture, eight; perforation of the stomach, one; and carcinoma in abberant mucosa, one). It is generally felt, therefore, that all patients who are acceptable risks, and who, in spite of medical treatment, have a symptomatic hiatus hernia should have surgery recommended to them.

We have had occasion to treat, surgically, twenty-five patients with symptomatic hiatus hernia.* From the symptoms that they present and from the findings at surgery, these patients may be divided into two groups. In the first group (Table I) are those patients who presented excruciating, deep, subxiphoid pain which bores directly through to the back or around both costal margins to the back. This pain appears suddenly,

Presented as an inaugural thesis before the Minneapolis Surgical Society, November 4, 1957.

*Several of these cases are presented with the cooperation of the St. Louis Park Medical Center, Minneapolis.

TABLE I. (GROUP I) PATIENTS WHOSE PREDOMINANT SYMPTOM WAS SUBXIPHOID PAIN

Pt.	Age	No. GI X-Rays	No. X-Ray Diag. Hiatus Hernia	Diameter Hiatus	Operation	Follow-Up
1. E.R.	57	1	1	5 cm.	H.H. repair	Well 6 yrs.
2. R.C.*	60	1	0	3 cm.	H.H. repair	Recurrence of pain after 1½ yrs. Reexplored. No hernia.
3. L.S.	24	2	0	3 cm.	H.H. repair	Well 3 yrs.
4. J.S.†	65	2	0(fundal mass)	6 cm.	H.H. repair	Well 2 yrs.
5. G.H.	23	2	0	3 cm.	H.H. repair	Well 1 yr. 10 mo. Questionable improvement.
6. P.B.*	34	2	0	4 cm.		Psychiatric care.
7. A.E.	54	1	1	3 cm.		Well 6 mo.

*R.C. and P.B. both had emotional disturbances, the severity of which was not recognized preoperatively. They should not have been selected for operation.
†The fundal mass seen on x-ray proved to be edema of gastric mucosa at site of herniation.

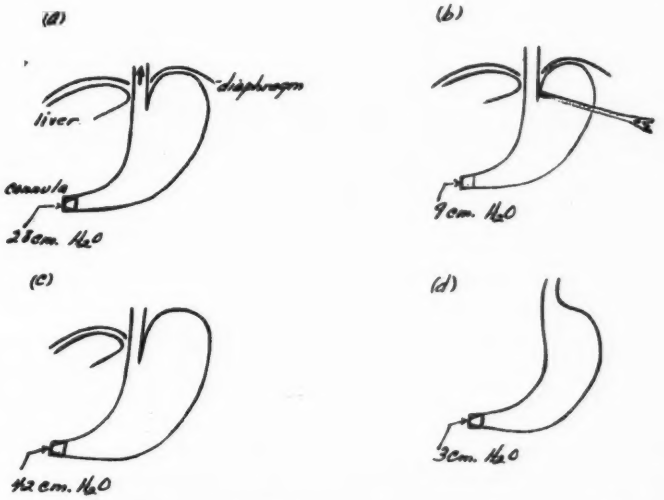


Fig. 1. Illustrates retrograde pressure necessary to overcome resistance of cardio-esophageal junction under varying conditions in young adult male cadavers. (From Marchand) (a) Control. Pyloris cannulated. Stomach undisturbed. (b) Clamp across stomach so as to exclude fundus. (c) Left leaf-diaphragm removed. Abdominal wall open. (d) Esophago-gastric angle abolished by freeing terminal portion and removing liver and right diaphragm.

without warning, persists steadily at maximum intensity for varying periods, and then suddenly and completely disappears. Except for moderate epigastric voluntary muscle-guarding during the attack of pain, the physical examination and all laboratory and x-ray studies are normal. This hernia is extremely difficult for the radiologist to demonstrate. We must, therefore, occasionally rely on the rather characteristic clinical picture to lead us to the diagnosis and to surgery as the only effective treatment. The clinician's failure to appreciate the radiologist's limitation in this area, may lead him to the unjust diagnosis of psychoneurosis or even to the removal of questionably diseased organs as a measure of desperation to bring relief to these patients.

Report of Case

Case 5 in Group I illustrates the foregoing. This young, stable farmer was first struck with severe subxiphoid pain radiating through to the back while sitting in church May, 1951, when he was nineteen years old. The pain lasted twenty minutes and was gone when the physician arrived. He subsequently had many similar attacks, which, as time went on, lasted longer and occurred more frequently. Evidence against this pain being functional in origin is the fact that occasionally as he was being driven to town to receive a hypo for the pain, he would get spontaneous relief, and then would turn around and return home to work. A gall bladder which concentrated well, but emptied slowly, and did not contain stones was removed on January 18, 1954. This did not relieve the attacks. An indication of the frequency of attacks is given by the fact that he received fifty-six hypodermic injections

TABLE II. (GROUP II) PATIENTS WHOSE PREDOMINANT SYMPTOM WAS REGURGITANT ESOPHAGITIS

Pt.	Age	No. GI X-Rays	No. X-Ray Diag. Hiatus Hernia	Diameter Hiatus	Operation	Follow-Up
1. R.B.	28	2	2	6 cm.	H.H. repair	Well 3 yrs. 7 mo.
2. E.H.	63	3	0	3 cm.	G.B. & H.H.	Well 3 yrs. 5 mo
3. R.J.	42	1	1	3 cm.	G.B. & H.H.	Well 3 yrs. 5 mo.
4. F.G.	69	1	0(Ready Regurg.)	4 cm.	Vagotomy Pyloromyotomy H.H. repair	Well 3 yrs. 4 mo.
5. J.N.	63	1	1	6 cm.	H.H. repair	Well 3 yrs. 2 mo.
6. C.B.	55	1	0	4 cm.	G.B. & H.H.	Well 3 yrs. 1 mo.
7. J.G.	64	1	1	8 cm.	H.H. repair	Well 2 yrs.
8. E.H.	59	1	0	6 cm.	H.H. repair C.B.D. stone	Well 1 yr. 10 mo.
9. D.G.	42	2	2	6 cm.	H.H. repair	Well 1 yr. 8 mo.
10. B.J.	38	2	0	3 cm.	G.B. & H.H.	Well 1 yr. 8 mo.
11. E.D.	45	4	1	7 cm.	H.H. repair	Well 1 yr. 6 mo.
12. E.O.	47	1	1	8 cm.	H.H. repair	Well 1 yr. 6 mo.
13. S.H.	65	2	2	10 cm.	H.H. repair	Well 1 yr. 5 mo.
14. H.T.	49	1	0	6 cm.	G.B. & H.H.	Well 1 yr.
15. A.S.	33	2	0	6 cm.	C.D. stone H.H. repair	Well 1 yr.
16. G.C.	62	1	1	10 cm.	G.B. & H.H.	Well 10 mo.
17. E.D.	58	1	1	5 cm.	H.H. repair	Well 9 mo.
18. A.N.	44	1	1	7 cm.	H.H. repair	Well 3 mo.

of either Morphine Sulfate grs. 1/6 or Demerol mgms. 100 from the time of onset in 1951 to December, 1955. At that time, after an extensive study had failed to reveal the diagnosis, an exploratory laparotomy was performed. Careful exploration revealed only a 3 centimeter hiatus hernia and absence of the gall bladder. The hernia was repaired and the patient has been asymptomatic since convalescence from this operation.

Case 3 in Group I presents an almost identical history. This young man had been hospitalized three times because of severe sub-xiphoid pain, and again extensive study did not reveal the diagnosis. He has been asymptomatic since simple closure of a small hiatus hernia defect.

Case 4 is of interest. This sixty-five-year-old man complained of attacks of midline epigastric pain. An x-ray of the stomach was interpreted as showing a mass in the fundus. This was confirmed on a repeat x-ray examination two days later. At laparotomy a hiatus hernia measuring 6 centimeters in transverse diameter was found. A generous gastrotomy was performed and showed only thickening of the stomach wall apparently due to edema from involvement in the hernia. Simple closure of the hernia defect has brought relief.

At operation in these Group I cases, the hiatus is usually found to admit two fingers into a preformed sac. The finding of a preformed peritoneal sac is of prime importance, since by exerting moderate pressure, one can normally insinuate two fingers into the esophageal hiatus. The pain associated with this small hiatus hernia is often sufficient to cause strong, stoical men to pale and perspire and can be best explained on the basis of visceral spasm involving the herniated portion of the stomach. The problem here is the simple mechanical one of herniation through an abdominal wall defect with which we are familiar in the case of inguinal hernia.

The symptoms and findings presented by the patients in the second group, with the possible exception of gastric ulcer, are due to reflux esophagitis. These symptoms and findings include retrosternal burning (heart burn), eructation, dysphagia, vomiting, flatulence, gastrointestinal bleeding, and esophageal stricture. In this group, the diagnosis is made by the radiologist somewhat more easily. We have, however, seen two patients (one of whom is included in this report) where it was possible to establish the diagnosis of hiatus hernia only after four separate gastrointestinal x-ray examinations. In several, it required two x-ray examinations to visualize the hernia. This is not surprising if we consider our experience with inguinal hernia, where we do not expect to demonstrate more than the very largest hernias at every examination. The symptoms of reflux esophagitis are easily elicited and are associated in the majority of cases with hiatus hernia even though such cannot always be demonstrated radiologically. In this group the esophageal hiatus, as examined at operation, tends to be slightly larger and frequently admits three fingers into a preformed sac.

In this second group (Table II), therefore, we have the dual problem of a diaphragmatic defect and incompetence at the esophago-gastric junction. It is felt that these problems are both mechanical. The experimental work referred to above suggests that the acute angle of insertion of the esophagus into the stomach is the factor of importance in maintaining the competence at this union. We have gathered an additional bit of circumstantial evidence to support this experimental study. First, we have found in careful dissection of the esoph-

ago-gastric area in autopsy specimens, that in the presence of a significant hiatus hernia, there tends to be effacement of the acute angle of insertion of the esophagus to the stomach as compared with

competent esophago-gastric union would give some meaning to the term "palliative resection" for carcinoma of the esophagus.

If we accept from the foregoing the explana-

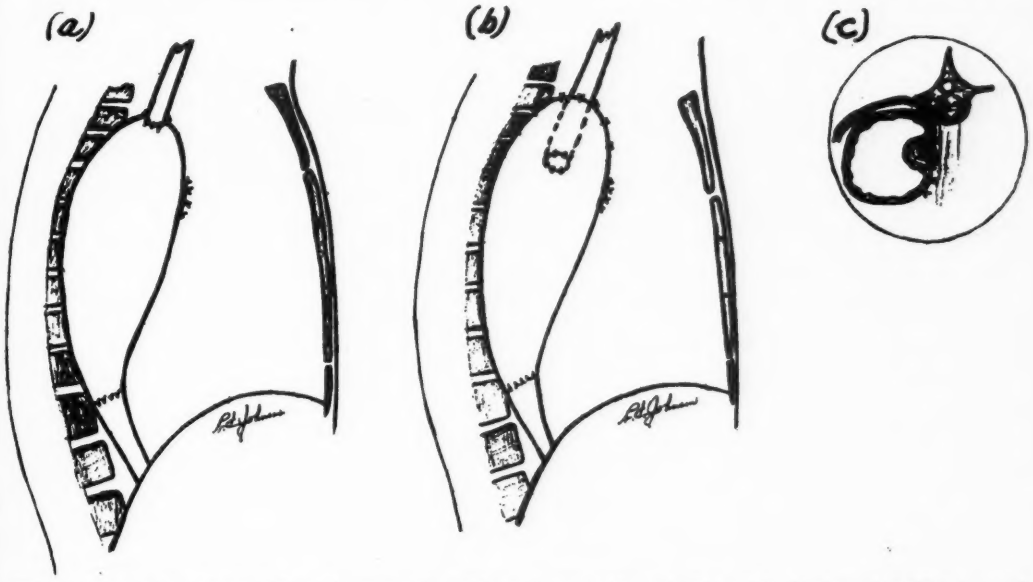


Fig. 2. Restoration of normal esophago-gastric angle and re-creation of the gastric fundus. (a) The type of thoracic esophago-gastrostomy usually pictured. (b) A thoracic esophago-gastrostomy which re-creates a gastric fundus and the acute angle of insertion of the esophagus into the stomach. (c) Horizontal section just above point of entrance of esophagus to stomach.

those specimens in which the esophageal hiatus was normal. Secondly, we have found that a real clinical test of the importance of the acute angle of esophago-gastric union in making this junction competent is presented by those patients who have had a resection of the esophagus. In five such patients an attempt was made to restore the normal esophago-gastric angle and re-create the gastric fundus (Fig. 2). This is accomplished by anastomosing the esophagus to the mediastinal side of the thoracic stomach approximately four centimeters caudad from the superior-most portion of the remaining stomach. The cephalad end of the stomach is then sutured in place to the mediastinum above the anastomosis to maintain it in the position of a gastric fundus. Post-operatively these patients have had no clinical symptoms of regurgitation and radiologically the union was competent in spite of the fact that it was now impossible for a "lower esophageal Sphincter" or "a diaphragmatic pinchcock" to function. These patients eat normally and sleep horizontally without fear of regurgitation and aspiration. Aside from the present report, a reconstruction which results in a

tion given for the symptoms presented by the Group I and II patients, our surgical objective is clear. In the Group I cases, where there is no symptom of esophago-gastric incompetence, the following procedure aimed at closure of the diaphragmatic defect suffices:

The hernia is reduced by placing traction on the stomach. The peritoneum overlying the esophagus is incised, and this structure with the accompanying vagus nerve is freed up. A penrose drain is passed about it to aid in retracting it inferiorly or from one side to the other as the margins of the hiatus are cleaned and delineated. The margins of the hiatus are then approximated posterior to the esophagus. Generous "bites" are taken on each side of the hiatus. This is especially important on the right where there is considerably less muscle mass in which to place the suture. Here the "bite" should extend well over toward the inferior vena cava, where the tissue is more fibrous and has greater tensile strength. The esophageal hiatus is closed sufficiently so that it will admit in addition to the esophagus and nasal tube, only the tip of the index finger. We have been unable to identify the

phrenico-esophageal ligament described by Allison. We do, however, attempt to maintain the esophagus in an abdominal position by incorporating in the most cephalad hiatal suture, the adventitia of the esophagus, posteriorly. Anteriorly, the esophageal adventitia is sutured to the anterior margin of the hiatus. Diaphragmatic contraction places no stress upon this repair, and there is, consequently, no need for right phrenic nerve interruption in these cases (Fig. 3).

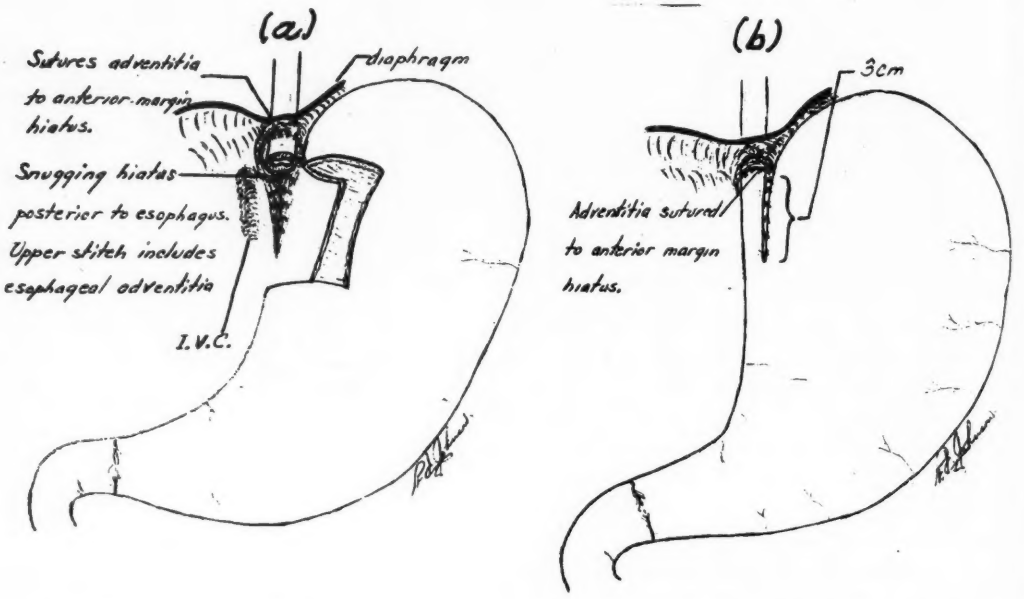


Fig. 3. Restoration of normal acute esophago-gastric angle providing a simple snug closure of the esophageal hiatus. (a) Lower esophagus and adjacent stomach cut away to show closure of hiatus. (b) Lower esophagus sutured to adjacent fundus to restore acute angle of union.

In the Group II cases where gastro-esophageal reflux is the primary problem, we must restore the normal acute angle of esophago-gastric union in addition to a simple snug closure of the esophageal hiatus. It is felt that this is best and most surely accomplished by adding to the operation described above, simple suture of the distal esophagus to the adjacent fundus for a distace of approximately three or four centimeters (Fig. 3). The results of this operation have been pleasing to the patient as well as to the surgon (Table II).

Mention should be made of left phrenic nerve paralysis in the surgical treatment of hiatus hernia. This measure is most likely to be effective in those cases with regurgitant esophagitis. There is a rational basis to be found in the work of Marchand for expecting this procedure to show a degree of success (Fig. 1). We are of the opinion, however, that it is rarely indicated in this or any other condition to interrupt the phrenic nerve and thus defunctionalize one of the most important muscles of the body. This applies particularly in the condition under discussion, where direct attack on the problem is simple, safe, and effective.

Summary and Conclusions

From a study of twenty-five patients with symptomatic hiatus hernia treated surgically, it is felt that patients with this disorder fall into two groups according to the symptoms they present. In the first group are those who have no regurgitant esophagitis, but are troubled primarily by attacks of severe upper abdominal pain thought to be due to visceral spasm involving the herniated portion of the stomach. In the second group are those patients whose primary difficulty is reflux esophagitis. In both instances the problem is a mechanical one which yields to a relatively simple procedure. The importance of the acute angle of esophago-gastric union in preventing gastro-

(Continued on Page 113)

Peripheral Neuritis

SIDNEY K. SHAPIRO, M.D.
Minneapolis, Minnesota

THE WORD "neuritis" often is used loosely and inaccurately as an elegant variation of the word "pain", particularly when such pain is of undetermined cause. The term "neuritis" is properly and explicitly restricted to designate the more or less prolonged disorders of sensation, motility and visceral function due to the disease or trauma of any peripheral somatic nerve or nerves.

A typical peripheral nerve is composed of motor, sensory and visceromotor fibers. The symptoms produced by the involvement of such a nerve will be due to involvement of the motor or the sensory, or the visceromotor components or various combinations of these three. It is thus possible to have a peripheral neuritis reflected as pure motor symptomatology such as is classically found in the wrist drop in lead poisoning, to have a peripheral neuritis with purely sensory symptomatology such as is frequently found in diabetic neuritis and to have pure visceromotor phenomena such as is encountered in incomplete injuries to the sciatic nerve. Any combination of involvement of the sensory, motor and visceromtor systems may exist and thus the picture presented by a peripheral neuritis may be very complex.

The symptomatology presented will depend upon the type of involvement of the peripheral nerve from which the patient is suffering. Each of the functions will be considered separately here, although, of course, any combination of the three can occur in the same individual.

Types of Involvement

Sensory Involvement.—The subjective symptomatology of which the patient may complain is extremely varied. He may complain of actual pain. However, frequently he complains of numbness and tingling. The patient may complain of paresthesias and finds it difficult to describe accurately the sensations which he is experiencing. Many compare these paresthesias when they involve the feet, for example, to walking on a thick rug. The patient may complain of hyperesthesia or increased perception of sensations. A common type of hyperesthesia encountered is the excessive pain elicited upon stroking the soles of the feet of a patient suffering from alcoholic peripheral neuritis.

The sensory system is composed of essentially two parts: (1) perception of deep sensibility and tactile discrimination and (2) the perception of pain, temperature and light touch. One sensory modality may be involved while the others remain intact. Involvement of pain or light touch can be detected, on careful examination, by using a pin and a wisp of cotton. The use of two test tubes, one filled with warm water and the other with cold water, will detect involvement of temperature. Deep sensation is determined by testing the position sense of the toes or fingers, by compressing tendon between the thumb and forefinger, by using a tuning fork to determine the patient's ability to perceive vibration sense, and by use of a divider with the points being placed at various distances apart and applied simultaneously to the skin. Joint pain is tested by attempting to move the joints through increased ranges of motion. Normally the patient will complain of pain or discomfort when the normal range of motion of the joint is exceeded but as in tabes dorsalis, it may be possible to far surpass this normal range of motion without any complaints on the part of the patient.

In neuritis, there may be great tenderness of the muscle supplied by the injured nerve, which may be sensitive not only to pressure but also to traction. Wartenberg emphasized that in every disease, the peripheral nerves respond with pain to brisk passive stretching and that this is a constant very early and very late sign of neuritis. Chronic dermatitis may occur in the area of cutaneous distribution of an injured nerve.

The velocity of transmission of nerve impulses is related to the caliber of the nerve fibers. On this basis, nerve fibers have been divided into

From the Division of Neurology, University of Minnesota Medical School, Minneapolis, Minnesota.

three groups: (A) touch, pressure and proprioceptor fibers with a diameter of 1 to 2 microns transmitting a velocity of 30 to 100 meters per second; (B) small myelinated temperature and localized pain fibers transmitting a velocity of 10 meters per second; and (C) unmyelinated diffuse pain and efferent vasomotor fibers transmitted at 1 to 2 meters per second. The rate of transmission averages 27 per cent less in the distal than in the proximal segments indicating that the velocity is closely related to the diameter of the fiber.

Motor Involvement.—The patient usually complains of weakness, clumsiness or inability to do some motor function which he could do previously without difficulty. On objective examination, weakness is demonstrated on testing isolated muscles or muscle groups. Usually the reflex is decreased or absent at the site of the weakness. On observation of the involved musculature, fasciculations may be visible. Partial interruption of a nerve may yield a tonic spasm of muscles which represents an irritative phenomenon.

Involvement of Visceromotor Fibers.—The peripheral nerves carry a varying complement of visceral components. In the median and sciatic nerves, this component is especially large. A burning type of pain is usually suggestive of lesions of these visceral fibers and it may occur independently of impairment of muscle power and cutaneous sensation. Vasomotor and trophic disorders (usually most marked peripherally) include objective warmth or coolness, rubor, pallor or cyanosis, hyperhidrosis or hypohidrosis, edema, atrophy of the skin and subcutaneous tissues, hyperkeratosis, pigmentation or depigmentation, irregular growth of hair and nails, and ulcers. The hair may actually fall out, in complete nerve injuries. With incomplete nerve lesions, there may be an excessive production of hair.

Special Testing of Peripheral Nerve Function

In most instances, by careful neurological examination, it is possible to determine the presence of neuritis. However, in some obscure problems, particularly where the differential diagnosis of hysteria or malingering is entertained, special testing of disturbance of nerve function may be of benefit.

These special methods include:

1. The determination of the electrical reactions of nerve and muscle. By the use of faradic and galvanic stimulation applied to the nerve and to the muscle, the presence or absence of a complete reaction of degeneration or partial reaction of degeneration can be determined.

2. The determination of chronaxie. The minimal strength of current of infinite duration necessary to give contraction is called the rheobasic voltage. The length of time the current twice this strength must flow to stimulate a nerve or muscle is called its chronaxie. Adrian has found that the chronaxie of a human muscle with an intact nerve supply is about 0.00016 second while the chronaxie of the muscle associated with the degenerated nerve is about 0.01 second.

3. Measurement of electrical resistance.

4. The application of paste of iodine and starch to the skin. This is sensitive indicator for the presence of perspiration.

5. The use of the plethysmograph, and the measurement of the local temperature of the skin. The retention or loss of vascular reaction when peripheral nerves have been injured can be inferred by measurement of the local temperature of the skin or by placement of a plethysmograph on the affected extremity. For example, if the median or ulnar nerves are paralyzed, the opposite extremity is plunged into ice water; there results no vascular response in the affected extremity but when the radial nerve alone is paralyzed, the vascular response is normal.

6. Histamine Test. Following the intradermal injection of 0.01 cc. or the application to a fine puncture wound of 1:1000 solution of histamine hydrochloride, normally a flare appears in three to five minutes. The flare is retained in hysterical anesthesia, or anesthesia of central origin. It appears also within the first six to eight days following interruption of a peripheral nerve; after this, however, the flare reaction does not appear in an area rendered anesthetic by a lesion of its nerve supply. As regeneration takes place, the histamine flare returns early.

7. Electromyography.

8. Examination of cerebrospinal fluid.

9. Roentgenograms of the extremities. There may be evidence of pronounced demineralization of bones when the extremity has been deprived of its nerve supply. The epiphyseal lead line may

be of great help in the diagnosis of lead poisoning in children.

Classification and Terminology

Various classifications have been proposed for classifying peripheral neuritis, according to: (1) Its clinical course; that is, acute, subacute or chronic. (2) The nature of the symptoms—referable to motor, mixed or sensory nerves. (3) The distribution of the condition, for example, mononeuritis, mononeuritis multiplex, multiple neuritis, polyneuritis or polyradiculitis. (4) The part of the nerve affected, i.e., radiculitis, ganglionitis, funiculitis, plexitis, neuronitis or peripheral neuritis. (5) The type of tissue affected—parenchymatous or interstitial. (6) Generalized polyneuritis or localized neuritis.

An etiological basis for the classification, however, appears to have the most practical significance and a classification based upon etiology is presented as follows:†

Classification of Neuritis

I. Toxic-Infectious
   1. Guillain-Barre's Syndrome
   2. Exogenous Toxins
      (a) Heavy metals—arsenic, lead, thallium, phosphorus, mercury, manganese, gold, silver, bismuth, copper, antimony (?), zinc (?), and tin (?).
      (b) Organic solvents—alcohol, carbon disulfide, benzene, tetrachlorethane, trichlorethylene, carbon tetrachloride, etc.
      (c) Carbon monoxide.
      (d) Miscellaneous—methyl bromide, triothocresyl phosphate (Jamaica ginger)
   3. Bacterial Toxins—diphtheria, botulinus.
   4. Serum Neuritis—tetanus antitoxin; pneumonia, meningococcus, scarlet fever, and diphtheria sera or antitoxin, antirabic treatment.
   5. Complicating Infections—mumps, scarlet fever, gonorrhea, typhoid fever, typhus fever, chorea, leprosy, measles, rabies, small pox vaccination, infectious hepatitis, and puerperal sepsis.
   6. Drugs
   7. Miscellaneous—insect bites, acrodynia, congenital pink feet, hereditary affliction of the feet, sun burn

II. Traumatic
   1. Fractures and dislocations
   2. Occupational trauma
   3. Trauma in athletics
   4. Trauma during operation or therapeutic procedures
   5. Birth trauma
   6. Trauma in war

III. Vascular
   1. Vascular occlusions due to—arteriosclerosis, mechanical compression (trauma, neoplasm, tourniquets, aneurysm, etc.), periarteritis nodosa, thromboangiitis obliterans, livedo reticularis, embolism, exposure to colds (radial nerve), reaction to radium.
   2. Vascular stasis (Volkmann's ischemia)
   3. Hemorrhage—purpura, hemophelia.

IV. Tumor
   1. Primary—neurofibroma, von Recklinghausen's disease, ganglioneuroma, amputation neuroma, neuromyoarterial glomus, Kaposi's disease, fusiform cysts or ganglions of the nerves, perineural cysts.
   2. Metastatic
   3. Ruptured intervertebral disc.

V. Metabolic
   1. Diabetes
   2. Pernicious anemia
   3. Porphyria
   4. Nutritional—beriberi, pellagra, gastric carcinoma, malfunctioning gastroenteric stoma, obstructive duodenal ulcer, gastric polyps, diaphragmatic hernia, intestinal fistula, prolonged vomiting and malnutrition.
   5. Neuritis of pregnancy.

VI. Hereditary
   1. Peroneal type of muscular atrophy of Charcot-Marie-Tooth (familial)
   2. Hypertrophic neuritis of Dejerine-Sottas (familial)

VII. Miscellaneous—scleroderma, disseminated lupus erythematosis, pregnancy, Boeck's sarcoid, acromegaly, radium, amyloid disease, gout (?).

Summary

1. Peripheral neuritis may present with motor, sensory or visceromotor symptomatology or various combinations of these three.
2. Features of the sensory, motor, and visceromotor involvement in peripheral neuritis are reviewed and the special testing of peripheral nerve function outlined.
3. A practical etiological basis for the classification of peripheral neuritis is presented.

1218 Medical Arts Building
Minneapolis 2, Minnesota

†The details of all the variations of the involvement in each of these conditions can be obtained by consulting any standard textbook in Neurology.

Use of Primacaine in Intraocular Ophthalmic Surgery

A Clinical Evaluation

HOWARD DINSDALE, M.D.
Saint Paul, Minnesota

A RELATIVELY new anesthetic agent, 2'-diethylaminoethyl-2-butoxy-3-aminobenzoate hydrochloride (Primacaine hydrochloride) has been studied by a group of investigators[1] and used for various purposes clinically in this country and Canada.[2] The author, under the supervision of Edward P. Burch, M.D.,* has recently conducted a clinical survey of Primacaine in intraocular ophthalmic surgery. No attempt has been made to compare it with xylocaine, procaine, and other anesthetics.

The patients represented a consecutive, unselected, series of intraocular cases operated upon by a group of local ophthalmologists. The patients varied in age from twenty-one to eighty-nine; the majority being fifty years of age or older. The series is comprised of eighty cases of cataract without glaucoma, two cases of cataract with glaucoma, ten cases of glaucoma, two cases of anterior chamber foreign body, and one case of cataract after-membrane. Preoperative sedation consisted of thorazine 25 mgm. or phenergan 50 mgm. with demerol 50 mgm. in patients under seventy years of age. In patients over seventy years of age, the dosage of both drugs was halved.

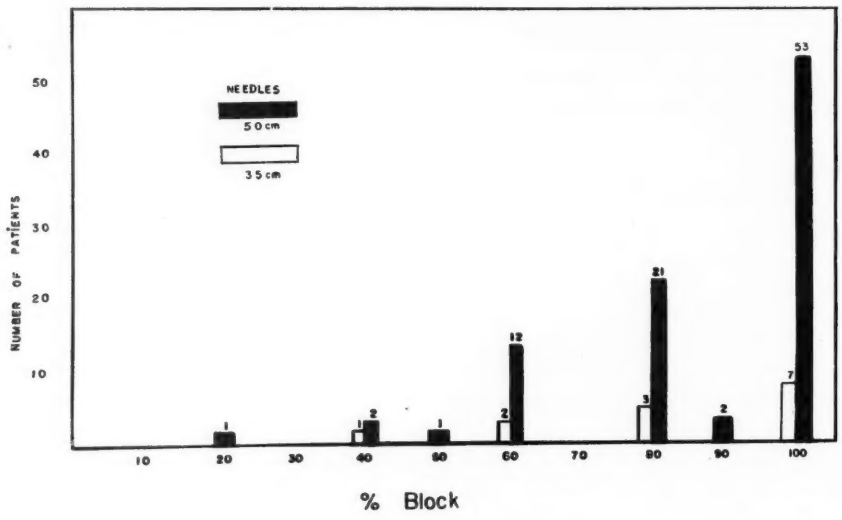


Fig. 1. Ocular akinesia.

The 5.0 cm. 25-gauge needle was used in all but a few cases. In these few cases (most of which were glaucoma) the 3.5 cm. 25-gauge needle was substituted. The trans-cutaneous method of retrobulbar injection and the Van Lint lid block were employed throughout. Primacaine hydrochloride with epinephrine 1:60,000 and hyaluronidase 5 viscosity-reducing units per cc. were used. Two cc. of solution were used for retrobulbar injection and 2.5 cc. for the lid block. The globe was massaged following injection for a period varying from three to five minutes.

The block was evaluated for ocular akinesia,

*Dr. Burch is Associate Professor of Ophthalmology, University of Minnesota, Minneapolis, Minnesota.

intraocular tension, lid akinesia, and patient comfort during the surgery. Ocular akinesia was evaluated on a percentage basis as advocated by Gifford. In this method 100 per cent indicates complete ocular akinesia, 20 per cent is deducted from 100 per cent for each rectus muscle still active, and 10 per cent for each oblique muscle still active. Intraocular tension was estimated as firm, moderately soft, and very soft. This was evaluated at the time of the corneal section on patients having no previous glaucoma. Firm tension was present when there was a definite tendency for iris prolapse and a lack of corneal wrinkling, moderately soft when there was corneal wrinkling without a tendency for iris prolapse, and very soft when there was corneal indentation. Complete lid akinesia was present when there was an absence of lid squeezing during and following surgery.

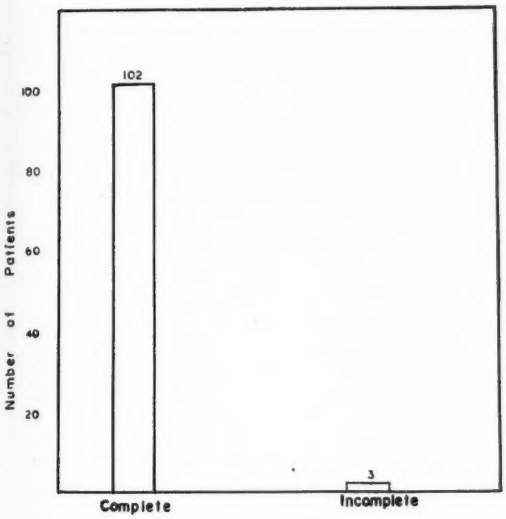


Fig. 2. Lid akinesia.

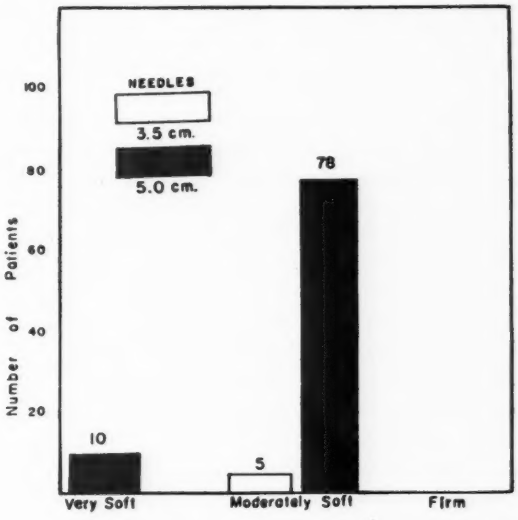


Fig. 3. Intraocular tension.

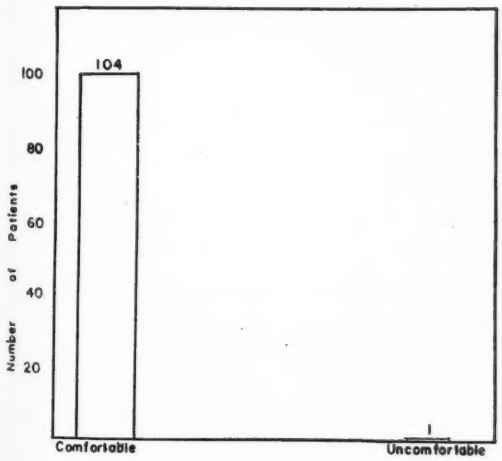


Fig. 4. Patient comfort during surgery.

Conclusion

In conclusion, the clinical results indicate that Primacaine hydrochloride represents a superior anesthetic agent for intraocular ophthalmic surgery.

References

1. Epstein, E., Meyer, M., and Ginsberg, H.: Experimental toxicity and local anesthetic activity of alkyl aminoethyl ester of meta-amino alkoxy benzoates; Current Researches in Anesthesia and Analgesia, 34:171-180 (May-June), 1955.
2. Rogers, M. A.: A clinical evaluation of primacaine hydrochloride. Modern Dentistry, 22:7-9 (July) 1955.

1508 Como Avenue
Saint Paul 13, Minn.

Hospital and Hospital Staff Responsibility for Maternal Mortality

D. W. FREEMAN, M.D.
ALEX BARNO, M.D.
Minneapolis, Minnesota

SUBSTANTIAL reductions in maternal mortality rates have occurred within recent years. Gross maternal mortality rates in Minnesota have decreased from 2.03 per 1,000 live births in 1941 to 0.49 per 1,000 live births in 1954, a reduction of 76 per cent. The proportion of preventable deaths decreased in the same period from 73.2 per cent to 30 per cent. In a recent five-year period in Minnesota, only 177 "obstetric" deaths occurred among almost 400,000 live births. A summary of these deaths showed, however, that 45 per cent were considered preventable.[1]

Clearly, room for improvement still exists. The responsibility of patients through failure to avail themselves of care, attempted self-induced abortions, suicides, etc., accounts for a negligible fraction of the deaths. This is in marked contrast to earlier studies such as the "New York Study" which showed that patients were responsible for over one-third of the deaths.[2] It follows that continuing efforts to reduce maternal mortality must be directed primarily to physicians doing obstetrics. This is now being done by education at medical school and at postgraduate levels, at medical society meetings, by publication of maternal mortality study committee findings, and by letters and case summaries directed to the physicians concerned. Since almost all of the deliveries are conducted in hospitals, maternal mortality rates could probably be lowered further by general adoption and enforcement of effective hospital regulations governing the conduct of obstetric practice. Formulation and enforcement of such regulations should be the joint responsibility of the hospital staffs, the hospital administrators and the official health agencies concerned.

The purpose of this paper is to analyze the preventable maternal deaths which occurred in Minnesota during the five-year period 1950-1954, with the aim of determining what improvement could have been made had the hospital practice of obstetrics been better organized.

TABLE I. RESPONSIBILITY FOR PREVENTABLE MATERNAL DEATHS OCCURRING IN MINNESOTA HOSPITALS 1950-1954

Responsible	Number	Per Cent
Physician	66	90.4
Patient	2	2.7
Nurse-anesthetist	2	2.7
Laboratory	2	2.7
Hospital administration	1	1.4
Total	73	99.9

TABLE II. CAUSES OF PREVENTABLE MATERNAL DEATHS OCCURRING IN MINNESOTA HOSPITALS 1950-1954

Cause of Death	Number	Per Cent
Hemorrhage	33	45.2
Toxemia	12	16.4
Infection	8	11.0
Anesthesia	7	9.6
Electrolyte imbalance	3	4.1
Air embolism	2	2.7
Incompatible blood	2	2.7
Heart disease	2	2.7
Diabetic acidosis	1	1.4
Pituitrin shock	1	1.4
Over transfusion	1	1.4
Unknown	1	1.4
Total	73	100.0

During the time period mentioned there were eighty-three maternal deaths in the state which were judged by the Maternal Mortality Committee of the Minnesota State Medical Association to have been preventable. Of these, four resulted from non-obstetric causes and six patients died at home. The remaining seventy-three deaths occurred in hospitals and of these, physicians were judged to have been responsible for sixty-six and the hospitals through nurse-anesthetist, laboratory and/or administrative error were directly responsible for five. The responsibility for two deaths was charged to the patients (Table I). The causes of the seventy-three deaths are indicated in Table II.

From the Minnesota Maternal Mortality Study Committee. Drs. Freeman and Barno are Obstetrician-Consultants for the Committee.

Presented at the meeting of the Minnesota Obstetrical and Gynecological Society, Saint Paul, November 30, 1957.

TABLE III. TABULATION OF DEATHS DUE IN LARGE PART TO INADEQUATE PATIENT STUDY

Cause of Death	Number
Hemorrhage	7
Infection	5
Anesthesia	5
Toxemia	4
Electrolyte imbalance	3
Incompatible blood	2
Heart disease	1
Diabetic acidosis	1
Unknown	1
Total	29*

*39.7 per cent of 73 preventable deaths occurring in hospitals.

TABLE IV. DEATHS DUE DIRECTLY TO FAULTY CONDUCT OF LABOR, DELIVERY AND EARLY PUERPERIUM

Cause of Death	Number
Improper anesthesia	7
Improper use of pituitary extract	7
Accouchement force	6
Inadequate observation of patient in labor and/or postpartum	5
Internal version	3
Inadequate pre delivery preparation	3
Total	31*

*42.5 per cent of 73 preventable deaths occurring in hospitals.

Fifty-four of the deaths occurred in out-state hospitals and nineteen in the four largest cities in the state. This distribution of the deaths is out of proportion to the distribution of births occurring in the state, these being divided approximately equally between the four largest cities taken together and the remainder of the state. Data previously published showed that the "physician responsibility maternal mortality rate" was two and one half times higher in rural than in metropolitan areas.[1] Obviously, some fundamental differences in the hospital practice of obstetrics exist in the two areas.

Factors which have been evaluated as to their influence on the occurrence of these seventy-three preventable deaths, include patient work-up (history, physical examination, pelvic mensuration, laboratory and x-ray studies), conduct of labor and delivery (including administration of anesthesia), and consultation. In many instances more than one of these factors were involved in a death. This overlap accounts for the fact that in Tables III, IV and VI, percentages total more than 100.

Patient Work-up

There were many deficiencies in the most basic patient work-up and observation. The effects of such deficiencies are partially shown in Table III. In twenty-nine instances, the entire hospital record consisted of scanty nurses' notes only, while another nineteen did not have a complete history and physical examination recorded. Only two anesthesia records were found in a series of seven anesthetic deaths. Consultation records and progress notes by the physicians were almost uniformly missing or deficient. In not a single case was the prenatal record made a part of the hospital chart. Pelvic measurements were almost never recorded.

TABLE V. CONSULTATIONS OBTAINED IN CASES OF PREVENTABLE MATERNAL DEATHS IN MINNESOTA HOSPITALS 1950-1954

	Number	Per Cent
None	37	50.7
Inadequate and/or delayed	30	41.1
Adequate	6	8.2
Total	73	100.0

TABLE VI. TABULATION OF CASES OF MATERNAL DEATHS IN WHICH LACK OF CONSULTATION PLAYED A MAJOR ROLE

Cause of Death	Number
Hemorrhage	27
Toxemia	5
Infection	5
Electrolyte imbalance	3
Heart disease	2
Anesthesia	2
Diabetes	1
Unknown	1
Total	46*

*63 per cent of 73 preventable deaths occurring in hospitals.

Some of the disasters resulting from the lack of simple studies are illustrated by the following cases. Four patients who ultimately died of the effects of anesthesia did so because pelvic measurements were inadequately evaluated. Two of these occurred at the time of cesarean sections which were done because of "outlet contraction" in one patient and breech presentation in the other. Neither simple clinical nor x-ray pelvimetry was done in these cases. The other two developed prolonged obstructed labors due to unrecognized pelvic contractions and died of the effects of general anesthesia and aspiration in association with difficult operative deliveries. A fifth patient whose pelvis was never adequately evaluated died of a ruptured uterus following internal version and breech extraction done because of questionable obstruction of labor.

A patient died of the effects of a ruptured

uterus after attempted internal version and breech extraction for obstructed labor. The obstruction was due to the presence of a pelvic tumor which was undetected because pelvic examination had never been done.

Another patient was hospitalized for fifteen days at four months gestation with abdominal pain, nausea and vomiting and other signs of an acute abdomen. Four days after discharge she was readmitted and she expired twelve hours later. Autopsy showed a ruptured appendiceal abscess. On neither admission was a history or physical examination recorded. No laboratory work was done except for one hemogram and a single urinalysis, and the single consultation obtained was delayed and inadequate.

Still another patient was hospitalized for a total of three weeks. She had been vomiting for six weeks. No history, physical examination or laboratory work was done and no consultation was obtained. The patient died of shock, dehydration and electrolyte imbalance at fifteen weeks gestation. Another patient known before pregnancy to have congenital heart disease was admitted for a period of four days with signs and symptoms typical of congestive failure. No history or physical was done, no x-ray studies, electrocardiagrams or consultation was obtained and no treatment directed at the congestive failure was given. The patient was readmitted four days later still in failure and she died seventeen hours after admission.

Lack of adequate laboratory and x-ray facilities or failure to obtain minimal laboratory studies when available, together with laboratory errors, were major factors in twelve deaths. Some examples included three patients who died of unrecognized electrolyte imbalance, none of whom had any pertinent laboratory work done. A known diabetic who died in diabetic coma after more than three days of hospitalization had no laboratory work done except for a single urinalysis. Another patient died of peritonitis and septicemia five days after classical cesarean section for obstructed labor. The hospital had no facilities whatever for bacteriological study. In one of the deaths due to obstructed labor, difficult midforceps extraction, general anesthesia and aspiration (previously referred to), the hospital x-ray department had predicted that there would be no dystocia. This conclusion was arrived at from study of a single simple flat film of the pelvis with no provision made for correcting for distortion. Two patients died from administration of incompatible blood as a result of laboratory typing errors.

Conduct of Labor and Delivery

Forty-two and one-half per cent of the deaths being considered could be directly attributed to faulty conduct of labor, delivery and early puerperium including improper administration of anesthesia (Table IV). Sixteen or almost one-half of all the deaths from hemorrhage resulted directly from procedures which would never be allowed in a well regulated obstetrical department. These included six who died of hemorrhage from lacerations following manual dilatation of the cervix, three from ruptured uteri caused by performance of unindicated internal version and extraction and seven who died when recognized safety rules regarding the use of pituitary extract were violated.

Five patients in the hemorrhage group died almost directly as the result of inadequate observation in labor and/or the postpartum period. The following example is illustrative of this group. The patient was delivered by an unindicated version, performed because of questionable obstruction. The pelvic capacity, however, had never been adequately evaluated. No blood pressures were taken during labor, during the operative procedure or postpartum. One hour postpartum, simple observation of the patient made it obvious that she was in shock. One pint of blood was given but the shock was never corrected and the patient died four hours postpartum.

Three deaths from hemorrhage occurred principally because of lack of preparation in advance, when hemorrhage could have been anticipated. In numerous other instances, this was a less direct cause. One of these patients was hospitalized for two weeks with pain, anemia, a history of a missed menstrual period and bloody vaginal discharge. The pre-operative diagnosis was never established and a laparotomy was done without intravenous fluids being started or any blood being crossmatched. The patient went into shock during surgery at which time an ectopic pregnancy was found. She died one hour later before any effective anti-shock therapy could be given. Another patient died in a large metropolitan hospital of hemorrhage at cesarean section. No blood had been crossmatched pre-operatively. Still another died of pastpartum hemorrhage due to uterine atony five hours after delivery. No blood was made ready beforehand although the patient was bleed-

ing excessively on hospital admission and a total of only 500 cc. of blood was given.

Proper supervision of anesthesia departments would probably have avoided all seven anesthetic deaths. Four of these resulted from spinal anesthesia, all as a consequence of administration of an overdose of the anesthetic agent. One patient died as a result of violation of safety rules regarding the administration of trichlorethylene and two others died of aspiration after being given general anesthesia in circumstances where this was obviously dangerous. Both patients had been fed and had vomited prior to administration of the anesthetic.

Consultation

Adequate consultation was obtained in only six cases, delayed and/or inadequate consultation in thirty and none at all in thirty-seven (Table V). The unavailability of consultation or, more often, the failure of physicians to call available consultation when indicated, constituted a major factor in no less than forty-six deaths (Table VI). Many examples can be given. A multipara known to have been delivered previously by classical cesarean section was admitted with complaints of abdominal pain and vaginal bleeding. She was in shock on admission with a blood pressure of 70/40. Fetal heart tones disappeared shortly after admission. Diagnoses considered were placenta praevia, appendicitis and placental abruption. The patient expired in shock after minimal anti-shock therapy, seven and one-half hours after hospital admission. Autopsy showed a ruptured cesarean section scar.

In another case, an inadequately qualified consultant was called to see a patient after she had been in hard second stage labor for four hours with no progress being made. Signs of uterine rupture appeared shortly following this. The patient was given subcutaneous pitocin and a difficult forceps extraction of a 6000 gram fetus was done. The patient expired shortly thereafter of shock proven at autopsy to have been due to undiagnosed ruptured uterus. Another patient was admitted at thirty-one-weeks gestation with severe hypertension, four plus albuminuria and epigastric pain, the significance of which was not understood. An unqualified consultant who also failed to appreciate the significance of the epigastric pain, advocated membrane rupture although the cervix was firm and closed. The patient developed convulsions and died undelivered three and one-half days after admission. Still another patient had her uterus incompletely emptied following incomplete abortion. The uterus was packed. Blood replacement was deficient and antibiotic therapy was hopelessly inadequate. She developed signs of peritonitis and probable septicemia and died after six days of hospitalization with no consultation having been obtained. Numerous other examples illustrating the critical effect of inadequate consultations could be cited.

Comment

It is evident that the hospitals and their organized staffs were in too many cases remiss in failing to insist upon adequate patient work-up including history, physical examination, basic laboratory and x-ray data and incorporation of prenatal records including pelvic measurements. Routine observation of patients during labor and delivery and in the early postpartum period was often deficient. The hospitals, for the most part, have failed to define and enforce adequate rules and regulations regarding the performance of unindicated operative deliveries and the improper use of oxytocics. Inadequate organization and supervision of the administration of anesthesia was a large factor. A simple rule limiting the maximum dose of an anesthetic agent to be used for spinal anesthesia in pregnancy is clearly indicated for all hospitals. Anesthetic records were seldom kept. Equipment for administration of oxygen and for aspiration was often not available. None of the patients was observed postpartum in "a recovery room" and trained personnel in general are not available for administration of obstetric anesthesia.

Failure to insist upon adequate consultation for such things as obstetric operations, prolonged labors, toxemia, hemorrhage, retention of the placenta and puerperal sepsis played a major role in 63 per cent of the deaths. The ready availability of consultation in the large cities is manifestly a factor in the great disparity in the maternal death rates between the rural and metropolitan areas of the state. Making available adequate consultation might be considered a worthwhile project for the State Health Department or the Minnesota Obstetrical and Gynecological Society. However, in addition to increasing the availability of consultation, it is clear that a fundamental change in attitude is required on the part of many physicians toward the calling of consultation. Fre-

quently, adequate consultation was available but was not called for fear of "loss of face" or because of pride, ignorance or overconfidence on the part of the physician. Adoption by all hospitals of a regulation making consultations mandatory for such things as operative deliveries, prolonged labors, all forms of obstetric hemorrhage or toxemia, and so forth, could make a very real contribution.

It seems obvious that one of the major factors in the disparity of maternal mortality rates between metropolitan and rural areas in the State of Minnesota is the difference that exists in the organization within hospitals and in the staff control of important features of their conduct of obstetrics. With a few exceptions, all of the metropolitan hospitals are departmentalized and adequately organized. The conduct of obstetrics is closely supervised. Consultations are made readily available and, more important perhaps, are required under certain definite conditions. Hospital regulations forbid such outmoded procedures as accouchement forcé. They clearly define safe methods of using pituitary extracts, require adequate patient work-ups, have better organized anesthesia departments, and so forth. The few metropolitan hospitals which have failed to conform to these standards have also been the largest contributors of preventable deaths in those areas. Of nineteen deaths occurring in the four largest cities in the state eight occurred in two hospitals.

Summary

A remarkable 76 per cent drop in maternal mortality rates was achieved in Minnesota between 1941 and 1954. This commendable improvement is probably the result of a number of factors such as improved undergraduate training of physicians, increasing postgraduate and refresher courses in obstetrics, widespread use of chemotherapeutic agents, and early and adequate blood replacement. It includes an increasing percentage of babies born in new, modernized, and better staffed hospitals, from 49 per cent in 1935 to 99.1 per cent in 1955. In addition, public health measures such as education of the public to the importance of early and adequate prenatal and obstetrical care, as well as maternal mortality surveys for the education of physicians, are other factors in the reduction of maternal mortality. Perhaps the greatest credit is due the physicians in the state, most of them general practitioners, who are actually doing the obstetrics.

Despite the great achievements of the recent past, it has been shown by the Maternal Mortality Committee's studies that room for improvement still exists. It is with this in mind that an analysis of seventy-three preventable maternal deaths occurring in hospitals in the State of Minnesota in the five year period of 1950-1954 is presented. An attempt is made to determine what improvement could have been made had the hospital practice of obstetrics been more adequately controlled. Forty-two and one-half per cent of the deaths could be directly attributed to faults in the conduct of labor, delivery and the early puerperium which should not be tolerated in well organized hospitals.

Patient work-up including such simple things as history, physical examination and basic laboratory and x-ray studies was deficient in the majority of cases. This failure was considered a definite contributing factor in 39.7 per cent of the total deaths. Adequate consultation was obtained in only 8 per cent of the cases and lack of such consultation was a major factor in 63 per cent of the deaths. It is concluded that in spite of continuing improvement, further strides can be made in reducing maternal mortality by properly organizing and supervising the hospital practice of obstetrics. The time has come for each hospital accepting obstetric patients to give this serious consideration.

Formulation of a detailed workable set of recommendations for the organization and regulation of obstetrical departments in all hospitals in the state should be undertaken by the State Health Department in co-operation with representatives of other interested groups. Such groups should include the state and local health departments, state and local medical societies, the state hospital association and state and local obstetrical societies.

References

1. Barno, Alex, Freeman, D. W., and Bellville, T. P.: Minnesota maternal mortality study—Five-year general summary, 1950-1954. Obst. & Gynec., 9:336, 1957.
2. Gladston, Iago: Maternal Deaths—The ways to prevention. The Commonwealth Fund, 1937.

4959 Excelsior Boulevard
Minneapolis 16

Discussion

A. B. Rosenfield, M.D.—In the discussion of the five-year summary of maternal mortality in Minnesota, presented by Doctors Freeman and Barno, I would like to consider first, the present situation; second, what has

been done to reduce maternal mortality; and finally what additional steps are necessary to further reduce mortality to the so-called irreducible minimum. It should be emphasized that great progress has been made in the field of obstetrical care. Due credit must be given to the general practitioners who provide most of the obstetrical care in Minnesota as well as to the obstetricians for the excellent record they have established. In 1941 when the Minnesota Maternal Mortality Study first began, the rate was 2.03 per 1,000 live births; in 1954, it was 0.49 per one thousand, a reduction of 76 per cent. During this same period the proportion of preventable deaths decreased from 73.2 per cent to 30 per cent, a 60 per cent reduction. During the five-year period of the study, 1950-54, almost 400,000 live births occurred with 177 maternal ("obstetrical") deaths, a rate of 0.44 per one thousand live births. This was among the lowest rates in the United States. Excellent as this record was, the important fact is that seventy-three deaths or 41 per cent were considered preventable by the committee and that the maternal mortality rate was more than two and one-half times higher in the rural than in metropolitan areas.

A brief résumé of the factors responsible for the preventable deaths previously discussed by Dr. Freeman includes:

Deficiencies in patient-work-up and observation as evidenced by inadequate medical records, inadequate evaluation of pelvic measurements, and failure to obtain laboratory studies.
Faulty conduct of labor and delivery.
Lack of adequate consultation.
Failure to define and enforce adequate rules and regulations regarding operative deliveries and use of oxytocics.
Lack of resuscitative equipment and unqualified anesthesia personnel.
Lack of adequate laboratory or x-ray services and facilities.

What has been done to reduce maternal mortality? The State Department of Health is legally responsible for the issuance of licenses to operate hospitals, sanatoriums, nursing homes and boarding care homes under certain rules and regulations. An advisory board of nine members makes recommendations to the State Board of Health and assists in the establishment of rules, regulations and standards. Included on this board are four members appointed by the Minnesota Hospital Association and two physicians appointed by the Minnesota State Medical Association.

The governing body of the hospital or the board of trustees is responsible for its management, control and operation. It appoints a hospital administrator and the medical staff and formulates the administrative policies of the hospital. The governing body is responsible for the physical plant, equipment, facilities, and the employment of personnel. The medical staff is responsible to the governing body of the hospital for the clinical and scientific work of the hospital and for advice regarding professional problems and policies. This is a part of the statutory regulations for the licensing and operation of hospitals. The enforcement of hospital regulations should therefore be the joint responsibility of the hospital board of trustees, the hospital administrator, the hospital medical staff and the State Health Department.

Metropolitan hospitals are departmentalized or completely organized. The obstetrical service is closely supervised. Consultations are readily available and are usually required under definite conditions. Outmoded procedures are prohibited. Adequate patient work-ups are required. There are adequate laboratory and X-ray services and well organized anesthesia departments. This, no doubt, explains why only 25 per cent of the preventable deaths occurred in the metropolitan areas.

During the past decade, there has been improved undergraduate training of physicians, increasing postgraduate and refresher courses in obstetrics for physicians and nurses, refresher courses for paramedical personnel, widespread use of chemotherapeutic agents, and availability of blood for replacement. Public health measures have included maternal mortality studies for the education of physicians and hospitals, as well as education of the public to the importance of early and adequate prenatal and obstetrical care and where it may be obtained.

In the out-state areas many hospitals have in the past been entirely inadequate in size, equipment, facilities and budget. During the past ten years the Hill-Burton federal hospital construction program has made possible the construction of forty-nine general hospitals with 2,237 beds in the rural area. In addition, approximately 2,000 more beds have been provided in modern facilities entirely at local expense. As a result, there has been marked improvement in facilities and equipment which has reduced the overcrowding.

In addition to the physical plant, however, another serious problem which must be met, is the provision for adequately trained hospital personnel. Such personnel are in short supply and are especially difficult to recruit for small out-state hospitals. To help meet this problem, the State Department of Health has been engaged in recruitment activities and regional refresher training courses since 1950. However, since the first of January, 1957, it has been possible to intensify efforts through a U. S. Public Health Service grant. The objective of this demonstration is to improve patient care services in Minnesota hospitals by raising the level of practice of paramedical personnel. Three avenues of approach are used: (1) recruitment of new people into the various health fields, (2) training of existing personnel, and (3) recruitment of inactive trained persons. This is being attempted in the fields of medical technology, anesthesia (nursing), dietetics, medical records, and physical and occupational therapy. The Study employs a staff of consultants in these fields who are visiting hospitals to assemble data to be used as a baseline. On the basis of these findings, there will be determined ways and methods to improve care being given to patients in Minnesota hospitals.

And now, what additional steps are necessary to further reduce maternal mortality? As a result of visits to about one-half of the hospitals in the State, the Hospital Services Demonstration Project makes the following recommendations:

Medical Records.—Every hospital should have a well organized medical staff. The staff and hospital administrator should be record-minded. The staff should appoint a medical records committee which meets regularly and has authority to promote good standards and to reject substandard records. There should be a rule regarding delinquent records. A qualified record librarian is essential for satisfactory medical records. It is important to note that the major responsibility for an adequate medical record rests with the attending physician and he assumes final responsibility for its completion and accuracy even though the task may have been performed by an intern or resident.

The medical staff should determine what constitutes a complete and satisfactory record rather than have each staff member make a personal decision. An adequate patient work-up should include the medical and obstetric history, physical examination, and basic laboratory x-ray studies. The Joint Commission on Accreditation of Hospitals requires a prenatal history and physical examination on all obstetrical patients. The physician's prenatal record including pelvic mensuration should, therefore, be incorporated into the hospital record.

The medical staff should establish rules and regulations defining the types of cases in which consultations are mandatory and should require that consultation reports include findings and recommendations and be written and signed by the consultant. According to the Standards for Hospital Accreditation, consultations are required in all cases of major surgery in which the patient is not a good surgical risk, in cases of obscure diagnosis or questionable treatment, in all first cesarean sections, sterilizations and curettages.

Recognizing that consultations are frequently difficult to obtain in out-state areas the State Department of Health is attempting to employ a well-trained and qualified obstetrician on a full-time basis to provide such consultations to physicians on request. If this is not possible, other approaches will be necessary. However, as Doctors Freeman and Barno have pointed out, the mere availability of consultation is not sufficient. There must be a willingness on the part of physicians to make use of consultation services as they become available. This is well illustrated by the autopsy service for maternal deaths available through the State Health Department where such local service is not readily available. During the past three years exactly three requests have been received for autopsies.

Anesthesia.—Every hospital should have qualified anesthetists. With the limited supply of medical anesthesiologists who are found almost entirely in the metropolitan areas, trained nurse anesthetists are necessary. Six small hospitals visited to date use chloroform for obstetrics, six have no gas machines or resuscitative equipment. The registered nurse on the obstetrical floor of many small hospitals gives the anesthetic, usually with no previous training and scanty current instruction. Frequently, the attending physician sets the regulators on the gas machine and the nurse holds the mask. This apparently meets hospital medical staff approval. There should be training centers set up for giving basic instructions to registered nurses who must give anesthesia to obstetrical patients or in emergencies. The use of regional and pudendal block by the physician would require only analgesia for delivery.

Every hospital should have modern anesthesia and resuscitation equipment and trained personnel to use it, including a relief anesthetist. Training in anesthesia is necessary in small hospitals. Staff conferences should include the nurse anesthetist so that she may feel herself to be a part of the surgical team. There should be regulations governing the giving of food or fluids to women in labor. The provision of adequate anesthesia equipment and trained staff is the final responsibility of the governing board; but the regulation of maximum dosages of an anesthetic agent for spinal anesthesia is the responsibility of the hospital staff, the staff physicians.

Qualification.—Medical technologists and technicians should be qualified. In-service training has been conducted for office, clinic and hospital technicians. Obstetrical patients should be typed early in pregnancy and if Rh negative they should be checked periodically for sensitization. If not possible locally, Rh titers can be done at most regional hospitals, at the Minneapolis War Memorial Blood Bank or at commercial laboratories. A copy of the patient's work-up done in the physician's office should be sent to the hospital when the patient is admitted. At least, the physician should notify the hospital laboratory of a possible Rh problem. When an emergency crossmatch is ordered, careful typing is necessary as well as a check for ABO incompatibility and possible Rh sensitization. Plasma should be available in an emergency when a quick crossmatch cannot be done. It can be stored at room temperature for one and one-half years. In case of drastic emergency when whole blood is necessary, low titered O negative blood may be given without crossmatching.

Summary

Let me quickly summarize the points I am trying to make: There are statutory hospital rules, regulations and standards, even though minimal and general. There are standards for hospital accreditation. There are specialized consultants and services available from the State Health Department. Studies of hospital facilities and services are in progress and recommendations for improvement are being developed. In-service training of medical and paramedical hospital staff is available. But, utilization of avaliable resources depends on hospital management—the governing body of the hospital and its administrator; and even more so, on the hospital medical staff. The State Health Department has legal authority to enforce regulations and standards. But the legal approach, while immediate, has no carry-over value. We, therefore, prefer to use the educational approach. It takes longer and is more difficult but it has more permanent carry-over values and thus is more desirable in the long run.

Our most effective public health programs have been the result of the joint cooperative effort of all agencies,

(Continued on Page 120)

False Albuminuria Due to Para-aminosalicylic Acid

EARL T. OPSTAD, M.D.
Minneapolis, Minnesota

SINCE 1946, with the addition of streptomycin to the therapeutic regimen, the prognosis of the tuberculous patient has steadily improved. This has been due almost entirely to long term chemotherapy combined with selective resection. It is now common for patients to continue taking this medication on a prescribed program a year or two after discharge from a sanatorium, not because the disease was particularly advanced or recalcitrant but because the overall results are definitely better.

PAS (para-aminosalicylic acid) is used in nearly all instances in combination with streptomycin, or isonicotinic acid hydrazide or both. PAS is taken orally in the dosage of about 12 gms. daily and is absorbed rapidly from the gastrointestinal tract. After a single dose of 4 gms. of the acid form, the maximum blood concentrations range from 3 to 12 mg./100 ml. within ninety to one hundred twenty minutes. The blood level rapidly falls within three to five hours. Most of the PAS is excreted in the urine; usually, between 80 to 85 per cent can be recovered in the urine within ten hours of administration. The rapid excretion results in high urinary concentrations (400-900 mg. per 100 c.c.) of the various forms —free and conjugated amines.

Because, in our judgment, many patients who have had superior results from the modern antituberculous therapy have an excellent prognosis, it was decided to accept the application for insurance of a young doctor who had been discharged from a tuberculosis sanatorium less than a year previously and who was still on anti-tuberculous chemotherapy (INH and PAS). When his urine was examined at our laboratory, it was found to contain 75 mg. of "albumin" per 100 c.c. of urine by the sulfosalicylic acid method when compared with the Kingsbury-Clarke Standards. Two additional samples verified the "albuminuria." This, however, could not be verified by the doctor applicant. It was then decided to mail a urine sample without adding our preservative tablet. This proved to be the key. Subsequently, urine samples from ten patients who were taking PAS at Glen Lake Sanatorium near Minneapolis were tested for albumin. When all were found to be negative, our preservative tablet was added to each specimen and allowed to stand overnight and again tested for albumin. All were positive for "albumin" in concentrations varying from 5 to 100 mg. per 100 cubic centimeters. Eight of the ten patients were also taking INH and two were taking streptomycin in addition to the PAS.

The preservative tablet used by our laboratory is made by Magar Chemicals Inc., and contains "benzoic acid, a trace of a mercuric compound (one mg. per tablet) and the gradual evolution of formaldehyde in amounts too small to affect the chemical tests."

It was then decided to ask the co-operation of Merck, Sharp & Dohme. Investigation by their Chemical Control Division confirmed the false albumin test. Although they did not determine the full composition of the preservative tablet nor investigate the exact mechanism of the interference they did establish that Sodium PAS plus benzoic acid does not give false albumin tests, whereas Sodium PAS plus formaldehyde yields an interference in the form of turbidity. Formaldehyde thus appears to play a role, but it was felt that the complete mechanism is more complex and might involve the mercuric compound as well. Since formaldehyde is not present in the preservative tablet *per se* but is gradually evolved, this would account for the delayed interference.

Because of the increasing number of people

(Continued on Page 120)

Dr. Opstad is Assistant Medical Director, Northwestern National Life Insurance Company, Minneapolis, Minnesota.

Delivered before the Medical Section of the American Life Convention, Lake Placid, New York, June 1, 1957, and to the Minnesota Trudeau Medical Society meeting at the Minnesota State Sanatorium, Ah-gwah-ching, August 10, 1957.

Case Presentation

Colorado Tick Fever

O. M. HEIBERG, M.D.
VERNON A. VIX, M.D.
Worthington, Minnesota

IN THESE days of widespread travel and rapid means of transportation, physicians are likely to encounter illnesses that originate in areas other than their own. Because these illnesses are not ordinarily encountered the diagnosis may be unnecessarily delayed. Colorado tick fever is just such an illness which is easily suspected clinically if one is aware of its existence, but to the unfamiliar it poses a difficult problem. An instance of Colorado tick fever was recently encountered which is being reported to alert other physicians to its existence. This illness is unusual in this section of the country and only recently was it included for the first time in a standard textbook of medicine.[1]

Case Report

J. S., a thirty-seven-year-old white man, a physician. vacationed in Scenic Park, Minnesota from June 2 through June 5, 1957. During this time ten to fifteen ticks were removed from his clothing, but to his knowledge he was not bitten. On June 7 he left Minnesota for Colorado, and on June 9 he parked his trailer house in the Rocky Mountain National Park in Colorado. During the day of June 10 the patient hiked about the mountains of this area. Approximately five ticks were removed from his clothing during the hike, and at about 10 P.M. that same day a partially embedded tick was removed from his abdomen without difficulty.

At about 9 P.M. on June 13, almost exactly three days after he observed the tick bite, malaise, chilliness and slight headache were noted. These symptoms rapidly increased in severity and within one hour a fever of 102° was noted along with shaking chills and a severe frontal and retrobulbar headache. The patient continued febrile until the afternoon of June 16 (about three days). At this time no symptoms other than some lassitude and easy fatigue were noted. The patient was first examined on June 15, at which time no abnormalities other than fever were noted. A blood count at that time disclosed a total white blood count of 3,200 with 54 per cent neutrophils, 34 per cent lymphocytes and 12 per cent monocytes.

The patient continued with some malaise and lassitude until about 6 p.m. on June 18, at which time chilliness and headache recurred. Within four hours these symptoms reached a severity similar to the original episode of June 13. The patient's fever remained almost constantly at about 102° until it returned to normal early in the morning of June 21. A blood count done on June 21 disclosed a white blood count of 1,600 with 41 per cent neutrophils, 49 per cent lymphocytes, and 10 per cent monocytes. This degree of leukopenia occasioned some alarm on the part of the physicians who were caring for the patient, and because of this he was admitted to the Worthington Municipal Hospital on June 21, 1957 for more complete evaluation.

Shortly after admission to the hospital the patient's clinical course was reviewed and a diagnosis of Colorado tick fever was suggested. The patient was afebrile throughout his four-day stay in the hospital. White blood counts obtained on June 23 and 24 were 2,200 and 3,250 respectively. Other laboratory data included absent antibodies for tularemia and brucellosis, and absent heterophile antibodies. Blood cultures were also negative. A blood sample obtained from the patient on the afternoon of June 21 was mailed to the Rocky Mountain Laboratory in Hamilton, Montana. From this specimen a pathogenic agent was isolated by the inoculation of suckling mice which was identified as Colorado tick fever. A rise in the neutralizing antibody titer for Colorado tick fever virus was demonstrated in subsequent blood samples.

For a period of four days following the last episode of fever, the patient experienced marked lassitude and was easily fatigued. He returned to work on June 26 but it was not until about June 30 that he felt entirely normal. A white blood count on July 22, 1957 was 5,350 with 59 per cent neutrophils, 32 per cent lymphocytes, 5 per cent eosinophils and 4 per cent monocytes.

Discussion

The diagnosis of the above case was suggested by the typical clinical course of Colorado tick fever. After an incubation period of four to six days the illness begins quite suddenly with chills, fever, headache, general aches and anorexia. These symptoms continue for about two days and are followed by a period of remission lasting several days. This period is followed by a relapse of the original symptoms which usually lasts for several days and is at times more severe and of longer

From the Worthington Clinic.

duration than the initial episode. At times variations are seen. Third relapses are known and a continuous single episode of fever lasting five to seven days is at times seen. Physical examination is not revealing except for occasional mild erythema of the skin and conjunctival injection. Clinically, Colorado tick fever is indistinguishable from dengue except for the occurrence of a rash in the latter illness.

The most important laboratory lead to the correct diagnosis is the constant leukopenia which is usually most marked during the second episode of fever. Counts of less than 2,000 are common. Definitive diagnosis can be made by the isolation of the specific virus from the blood of the patient or the demonstration of the development of antibodies. The virus is isolated by injecting suspected blood intraperitoneally into three- to four-day-old mice.[2] For this purpose, mailed blood specimens are satisfactory, and the virus has been isolated from blood obtained anytime within the period of fever.

Thus far Dermacentor andersoni has been the only tick definitely shown to be the vector for this illness.[2] However, there are some reports of the virus isolated from ticks as far distant from the natural habitat of Dermacentor andersoni as Long Island, New York. To date, all instances of the illness have originated in the specific region to which Dermacentor andersoni is confined. The case presented no doubt originated in Colorado since Dermacentor andersoni is not found in Minnesota and the interval of onset after exposure to ticks in Minnesota was in excess of the known incubation of Colorado tick fever.

Summary

A case of Colorado tick fever has been presented which was recognized by a typical diphasic febrile course and leukopenia. The diagnosis was confirmed by the isolation of the specific virus from the blood of the patient.

References

1. Cecil, R. L., and Loeb, R. F.: Textbook of Medicine. Ninth Edition. Philadelphia: W. B. Saunders Company, 1955.
2. Eklund, C. M., Kohls, G. M., and Brennan, J. M.: Distribution of Colorado tick fever and virus-carrying ticks. J.A.M.A., 157:335, 1955.

SURGICAL TREATMENT OF ESOPHAGEAL HIATUS HERNIA

(Continued from Page 98)

esophageal reflux is emphasized by the result in five cases of esophageal resection where an attempt was made to restore this angle. In occasional instances where radiologic demonstrations of a hiatus hernia is difficult, it may be necessary to rely on the history to lead us to the correct diagnosis and proper treatment.

References

1. Breitner, B.: (Quoted by M. Ritvo: J.A.M.A., 94: 15-21, 1930.
2. Akerlund, A.: Diaphragmatic hernia of esophageal hiatus from anatomic and roentgenologic viewpoint. (In German). Acta radiol., 6:3-22, 1926.
3. Harrington, S.: Diaphragmatic hernia symptoms and surgical treatment in sixty cases. J.A.M.A., 101:987-994, 1933.
4. Merindino, K. A., Varco, R. L., and Wangensteen, O. H.: Displacement of the esophagus into a new diaphragmatic orifice in the repair of paraesophageal and esophageal hiatus hernia. Ann. Surg., 129:185, 1947.
5. Allison, P. R.: Reflex esophagitis sliding hiatal hernia and the anatomy of repair. Surg. Gynec. & Obst., 92:419-431, 1951.
6. Ellis, F. H., Jr.: Experimental aspects of the surgical treatment of reflex esophagitis and esophageal stricture. Ann. Surg., 143: 465-470, 1956.
7. Wangensteen, O. H., and Leven, N. L.: Gastric resection for esophagitis and stricture of acid peptic origin. Surg. Gynec. & Obst., 88:560-570, 1949.
8. MacLean, L., and Wangensteen, O. H.: The surgical treatment of esophageal stricture. Surg. Gynec. & Obst., 103:5-14, 1956.
9. Jackson, Chevalier: Diaphragmatic pinchcock, so-called "cardiospasm." Laryngoscope, 32:139, 1922.
10. Lerche, W.: The Esophagus and Pharynx in Action. Springfield, Illinois: Charles C Thomas, 1950.
11. Lendrum, F. C.: The anatomic features of the cardiac orifice of the stomach. Arch. Int. Med., 59:474, 1937.
12. Marchand, P.: The Gastro-esophageal sphincter and the mechanism of regurgitation. Brit. Jour. Surg., 42:504-513, 1955.
13. Blades, B., and Hall, E. R.: The consequences of neglected hiatal hernias. Ann. Surg., 143:822-832, 1956.

829 Medical Arts Bldg.
Minneapolis 2, Minnesota

Continuation Study

Expected Behavior in Children

LEE FORREST HILL, M.D.
Des Moines, Iowa

CERTAIN patterns of behavior occur with such regularity at different age periods in infants and children that they may be regarded as normal or expected. Yet such patterns of behavior are frequently of concern to parents and lead to parent-child conflicts which could be avoided with better understanding. The specific items selected for discussion are those which experience has shown to be most frequently bothersome to parents.

No two children are exactly alike. Even identical twins have some distinguishing features. On the other hand, it is equally true, as has been shown by Gesell and others, that within certain individual variations, all normal children pass through much the same maturational sequences in growth and development from birth to adult life. It is customary to classify growth and development under four broad categories—physical, intellectual, emotional and social. The maturational sequences of physical and intellectual growth are readily understood by everyone. Thus the child sits before he stands, stands before he walks and walks before he runs, and he progresses in height and weight from year to year. Likewise, he begins his intellectual development with his first smile at around six weeks of age, reaches for objects in his vicinity by five or six months, says a few words shortly after a year of age, talks pretty well by two and begins to read in the first grade of school. To expect any different sequences of events in these categories of growth and development would, even to parents, be ridiculous. But the behavior patterns of emotional and social development, while equally characteristic in the different age periods are less obvious and therefore less readily accepted. For instance, it is a little difficult for parents to be sanguine about such behavior as temper tantrums, negativism and curiosity about how the opposite sex is constructed—especially when the latter leads to investigation with the neighbor's child. When the four-year-old calls his mother "an old dummy" or tells her to "shut up," it takes a good deal of persuasion to convince her to ignore such language on the basis that "that's the way four-year-olds behave, and it's but a passing phase."

Physicians whose practices embrace the care of children have an opportunity to add greatly to the day-to-day satisfaction and pleasure parents have in living with their children by anticipating for them and interpreting to them expected patterns of behavior as the child proceeds from one age period to another. It is to be hoped, too, that such an approach might lessen the chances of later development of serious behavior problems which will require the services of the psychiatrist to straighten out. It has been well said that most of the problems parents have with their children arise from the fact that children behave as children. Too often parents expect adult behavior at an age when it is impossible of achievement, and they feel thwarted and incompetent by their failures.

Let us consider some of the expected types of behavior in the first two years of life, which is a most important period for the establishment of good parent-child relationships, and let us begin with the crying infant. Why do infants cry? What is meant by "three months colic?" Among the many suggested causes are swallowed air; allergy to milk, orange juice, or fish oil; hunger; excessive fat or carbohydrate in the formula; too small hole in the nipple in bottle-fed infants; enterospasm; "imbalance of the autonomic nervous system"; lack of parental affection; and a host of others. Brennemann, in about 1935, stated that "colic" is due to an "excessive accumulation of gas in the intestines fostered by the highly fermentable content of breast milk" and that, as a result, "the over-distended intestines become kinked and acutely obstructed at the bends." Attempts to overcome this obstruction, he stated, give rise to colic in the same manner as pain is produced by obstruction in a ureter or in a bile duct. It is doubtful

Presented at the Center for Continuation Study, University of Minnesota, March 19, 1957.

if many today would agree with this view of Brennemann's.

On the other hand, "full of gas" and "gas on the stomach" are common complaints voiced by parents about this period of infancy. That its source is largely swallowed air can be demonstrated very simply. An x-ray film ten to twenty minutes after birth will show the stomach already distended with air. Two hours later the air will have progressed through most of the small intestine and by six or seven hours will have reached the rectum. Thus attempts to rid the baby of gas are bound to be futile, since obviously there is no such thing as a "gas-less" baby.

Aldrich studied infantile colic as one of his first projects when he went to the Mayo Clinic in charge of the Community Child Health Research Project. Fifty babies in the newborn nursery were observed day and night for eight days. Observations were kept on the total number of crying episodes, the duration of each crying period, and the time of day when the crying occurred. As nearly as possible the cause of each crying episode was recorded. An average of eleven crying episodes per baby occurred per twenty-four hours. The longest period was four hours, and the shortest three-quarters of an hour, with an average of two hours per baby per day. The peaks of crying came at 6:00 P.M. and at 12:00 midnight when there was a minimum of nursing service available. In a subsequent study, Aldrich reported that increasing the personnel at these periods reduced the crying by fifty per cent. Thirty-five per cent of the babies cried because they were hungry, 10 per cent because they had soiled diapers, and 20 per cent because their diapers were wet. In 35 per cent no apparent cause for the crying could be ascribed. Aldrich speculated that the changed environment from intra-uterine to extra-uterine existence played a part. In the uterus was darkness, quiet, and a soothing rocking motion as the mother moved about. Externally there was noise, light, clothing, changing temperature, and lack of rocking.

Most practitioners would probably believe that babies really start to cry after they go home. In a study in homes using mothers as observers, however, Aldrich found there were only four crying episodes per day on the average, as compared to the eleven in the hospital. One inference to be drawn from these studies is that crying is directly related to the amount of attention received by the baby.

Whatever the cause of crying, or "three months colic," it is an extremely disturbing factor to parents, particularly to those with their first-born child. What is to be done about it other than frequent formula changing or giving a sedative drug such as phenobarbital? Several possibilities must be mentioned. First of all, one must understand that crying in the infant in the first few months of life is a protective reflex designed by nature to call attention to the infant's needs at a time when it lacks other means of making its wants known. It is a disappearing reflex, for, as cortical development takes over and the baby has other means of expressing its needs, the crying reflex gradually disappears. This protective reflex is an extremely wise provision on the part of nature. One wonders what the chances would have been for survival of the human race without it. Crying in the young infant, then, may be thought of as expected charactristic behavior for this period, just as there are other characteristic patterns of behavior in other periods of childhood.

Time should be taken by the physician sometime during the mother's pregnancy preferably in parents' classes or during the postpartum hospital period, to educate the parents to expect that the baby will cry when they go home. It is generally accepted, at least among the laity, that subsequent babies are more easily managed than first babies. There is, of course, no difference in the babies, but it is the parents who have changed. Having been through the experience once, they no longer become panicky every time the baby cries. Crying and "colic" are seldom troublesome factors in the home when there is an experienced and competent baby nurse in charge or when the baby is in the hospital attended by skilled nurses. Certainly, any baby that cries excessively should be comforted. An intelligent search for the cause should be made. It would be tragic to overlook an incarcerated inguinal hernia, an intussusception, or an abscessed ear in a crying infant on the assumption that its crying was due to some vague disturbance called "colic." If hungry, the baby should be fed, even though it may not be the scheduled time. If the whole feeding is gulped down it should be given more and the formula subsequently strengthened if need be.

When there is a reasonable chance that the crying may be due to milk allergy, the baby should be given a milk substitute on a trial basis. In general, however, frequent changing of formulas

is unlikely to improve the situation. Certainly serious thought should be given before taking a thriving baby off the breast merely because it is crying.

By the end of three months most parents have become accustomed to the task of raising a baby, and the period of crying or "three months colic" has come to an end. The remainder of the first year is usually rather peaceful and pleasant. Only a few minor things arise to disturb the equanimity of the parents. One of these is thumb sucking, which is of such common occurrence in infants that is may be classed as a normal behavior trait. "But won't it deform the baby's jaws?" is the parental complaint. They can be assured that if the practice stops by four or five years of age, as it practically always does simply because the child has matured sufficiently to put infantile methods of behavior behind him, the jaws will in the next few years of development resume their normal contours. A comparable example is seen in the spontaneous straightening of the legs by six or seven years of age when they were bowed or knock-kneed at two. The psychological effect or yanking the infant's thumb out of his mouth can be brought home rather sharply by suggesting to the mother that she yank the cigarette out of the mouth of her unsuspecting husband every time he starts to smoke. A little time out may well be taken to explain to parents that the habit children have of putting everything into their mouths may at one time have served a useful purpose. It is conceivable that in the ancient struggle for existence life itself may have depended upon the chance that what was put in the mouth might have some nutritional value. Such an explanation as this seems preferable to a Freudian concept of sexual gratification.

Somewhere around six months of age, most infants will exhibit a fear of strangers. Mother is likely to be apologetic for her infant's behavior, and explain that she has not had him out among people very much. Again, she needs to be reassured that such behavior is quite normal. In fact, it is a useful index of normal intelligence, for mentally deficient children are unable to differentiate between familiar features and those of strangers. By ten or twelve months of age most infants will have matured sufficiently no longer to be afraid of strangers unless advances are made. If one is imaginative, it is not too difficult to speculate that "fear of strangers" is a protective reaction that at one time served a useful purpose. When danger threatened in the appearance of something unfamiliar, such as a roaming forest animal in search of lunch, the lusty howl of the threatened infant quickly brought father with his stone axe to the rescue.

About the end of the first year infants develop the habit of body curiosity. They poke their fingers into their ears and navels, and girls into their vaginas, while boys handle their genitals. Sometimes girls sit with one finger poked into the vagina while twisting their hair with the other hand or sucking a thumb. Mother does not mind the ear-fingering very much, although practically all mothers at one time or another request that the ears be examined. Nor does she object particularly to explorations of the navel with the examining finger, but she can no longer restrain herself when the curiosity extends to the genitals. It may or may not help to point out to her that so far as the baby is concerned there is no difference between the genitals and the ear. The sex idea is in the adult mind, not in the infant's. The stage of body curiosity will pass whether mother does anything about it or not, but it is best to ignore the whole thing.

When the second year approaches, a new set of circumstances arises which makes the relative tranquility of the first year seem mild indeed. Now cortical development has proceeded to the point where the infant has ideas of his own. Moreover, he has developed the motor ability to enable him to attempt to put his ideas into effect. He has now become a personality in the family whose behavior and actions have to be reckoned with. Whether the family is to live in comparative peace or turmoil depends upon how situations are handled. The second year is one of the most, if not the most, important of all the formative years. The physician may well spend half an hour sometime between the tenth and twelfth month with parents, apprising them of what lies ahead.

The conversation goes something like this: "You've had your easiest year. You could put the baby in his bed or in his play pen and he'd stay there. Feeding hasn't been much of a problem. But in the year ahead of you, you're going to be confronted with an entirely different situation. It is a year when the baby will be forming many fundamental health or behavior attitudes. You'd like them to be good ones so you can live with him pleasantly, but whether they are good or bad

depends in large measure upon how you manage him. I should like to have you think of yourself as a teacher as well as a mother in your approach to the tasks that lie before you. The school teacher achieves her goals of teaching children to read, write, and spell by pleasantly conducted daily lessons. She doesn't expect the child to learn to read the first time a book is presented to him. Nor does she do the reading for the child. Instead, she helps him as much as is necessary, which is quite a bit at first, but with gradual shifting over to the child as his capabilities increase until finally he goes pretty much on his own. Instead of book-learning you are going to teach behavior attitudes, but you go at them in the same way. Now what are these behavior attitudes with which you'll be concerned during this next year? Briefly, they are eating, sleeping, play, temper-tantrums, training. Let me tell you a little about each of them."

Sometime around the end of the first year it may be expected that most children will slow up in their intake of food. The reason for this is that the rapid growth of the first year has come to an end. Average weight gains for the first year of life are approximately fourteen to fifteen pounds. During the second year the gain drops to around five or six pounds. Obviously, less food is required when weight gain is slow than when it is rapid. Parents who are unaware of these facts become concerned about the decreased food intake and frequently resort to various subterfuges to persuade the child to eat, not what he wants, but what they want. Not infrequently this marks the beginning of an eating hassle between parents and child which may last throughout childhood until the rapid growth spurt of puberty is reached, and the child again consumes large quantities of food. Most of this unpleasantness could be avoided by some timely advice from the physician in advance. When parents are expecting the decrease in food consumption, they can accept it calmly. They should be told that under no circumstances are they to urge, coax, or force food. It should merely be presented to the child and if he turns his head away, the spoon should quietly be placed back in the dish. If after a few more trials it becomes clear the child is not interested, the meal is terminated pleasantly and with no show of emotion. To eat when one is hungry is a pleasant experience, but to be forced to eat when one is not hungry is unpleasant, and in children usually leads to conditioning against the spoon and the whole performance of being fed.

It should be stressed, too, that most infants somewhere around a year of age make their first attempts to feed themselves. These attempts should be encouraged, not discouraged by keeping the food out of the child's reach, or by tying up his hands so the mother can have an unimpeded pathway to his mouth. Food ends up in the child's hair and is spilled on the floor, but some of it gets to his mouth. With a little experience and help from his mother, he will improve, and before long he will graduate to a spoon because it works better. Many children are capable of feeding themselves completely by thirteen or fourteen months of age, and all of them by eighteen months, provided the mother does a proper job of teaching. Mothers who have not been informed about the importance of teaching children to feed themselves at an age when they are capable of doing so are likely to insist upon doing the feeding themselves because it is less messy, and they can get through the task more quickly. Once children have acquired the ability to feed themselves, they should be expected to do so from then on. No eating problem can arise so long as the parents refrain from interfering. Some years ago, approximately 35 per cent of the preschool children brought for consultation came with the complaint "My child won't eat." What was really meant was "My child won't eat what *I* want him to." Today, eating is seldom a problem among the children whose parents have been educated in advance. Parents should remember that children are naturally dawdlers and should not expect them to attack their food with the gusto of a farm hand. It should be remembered also that many children do not want or require the three meals a day characteristic of adult eating. Many eat heartily only once a day, others may eat small amounts for two or three days, then eat several heavy meals, and some may eat lightly for a week or so only to make up for it in the next two or three days. Whatever the pattern of eating, it is interesting to observe that children who are permitted to run their own eating affairs maintain their positions on their growth charts with surprising constancy, despite the parents' protestations that "he doesn't eat a thing."

Eating is one of the commonest problems parents have with their children. In normal children,

in most instances, it is an unnecessary cause of parent-child conflict.

Sleep disturbances are another common problem. Some children have never learned to sleep well at night, dating perhaps from the period of three month's colic when a sleep resistance habit became rather firmly entrenched. There are many others, however, who have slept well throughout the first year but who at about one year of age raise a fuss about going to sleep. Frequently this coincides with the child's newly acquired ability to pull himself to his feet. Whereas formerly, when put to bed, he was content to lie quietly he now pulls himself to his feet, shakes the side of the bed vigorously and demands to be taken back into the family circle. How the parents meet this situation the first time may have much to do with the sleeping pattern for many months. If the child is picked up, rocked, fed or put in bed with the parents, a habit is established and it may be expected that the performance will be repeated nightly, and perhaps many times throughout the night. One harassed mother had been up with her two-year-old six and eight times every night for a full year, and the habit had started under the exact circumstances described above.

The answer is not to be found in sleeping bags, restraining jackets, or tying the child down in bed, but rather in teaching him that once he is put to bed it is expected that he will stay there during the night. The mother should, of course, go to him at once, first, to make sure that he is not in trouble and second, to prevent fears or anxiety about darkness or being left alone. He should be left in his bed, however, with firm instructions to lie down and go to sleep, accompanied by a motherly pet on the head or even a hug and a kiss. Then the mother leaves. If the commotion continues, she returns in ten or fifteen minutes and repeats her admonitions, but perhaps in a little firmer tone. This may have to be repeated several times, but the mother should emerge the victor. Managed in this way, the usual outcome is that the child, after a few nights, abandons the struggle and acquires an acceptable sleeping habit. Here again is illustrated the importance of teaching the desired behavior rather than resorting to punishings or sedative drugs.

A third pattern of expected behavior about which parents may well be given some advance information is the tendency most children have, after they have acquired the ability to walk, to get into things. In their explorations they flit from object to object pausing only briefly to familiarize themselves with something new. They climb anything climbable and handle anything available. They cannot be expected to know that things will break, that they may be expensive, or that they are dangerous. Adults should know that the child in this period has the whole world of things and of people before him about which he has to learn.

What may one do with him during his waking hours now that he has outgrown his play pen? There are two choices: let him have the freedom of the house, or assign him a limited area such as a room with a gate at the door. Some parents take the position that they are going to have things in their home just as they were before the baby came and that they are going to teach him to leave things alone. This seems unwise. Perhaps it can be done, but if so there will need to be such constant conflict between the child and mother that her "no" eventually is likely to convey little meaning because it has been used so often. If the child is to be given free run of the house, the least that might be done to lessen conflict is to remove temptation by putting out of his reach those things which he is not to handle.

A far better arrangement is providing the child a place to play downstairs, such as a room with a gate at the door, where everything is his to do with as he wishes. Parent-child conflict is avoided, mother can go about her work in peace, and yet the open door provides an opportunity for social contact between them. An additional advantage is that this arrangement reduces the risks of accidents, both from trauma and from poisons. It is to be hoped that the physician in his interview at this age period will stress to the mother the necessity of checking her household to make sure that hazards are reduced to the minimum.

Children who are taught from the beginning to be contented in a restricted and safe play area, particularly in the forenoon when mother has work to do, make life much more pleasant for both mother and child. In the afternoon the child is entitled to a tour of exploration about the house or out of doors so that his education will not be neglected.

Temper-tantrums also have their beginning around the first year of life and are to be expected for a couple of years thereafter. Children and adults have the same emotional set-up, but it takes a greater stimulus to call forth an explosion of

temper in an adult. The infant has a temper-tantrum over a minor stimulus such as inability to accomplish something he is trying to do or upon being restrained from carrying out some project he has in mind. The treatment is not to throw a bucket of cold water on him, to spank him, to shut him up in a closet, or even to put him to bed, all of which add to his emotional upset. Rather should the storm be ignored, or if the task he has been attempting to perform has proved too difficult, a friendly helping hand may be proffered. The outcome to be avoided is the continuation of temper-tantrums into an age period when they normally should have ceased, because the child has found that his behavior is an effective method of gaining a desired goal. Calmness and aloofness until the storm is over should be the mother's attitude, to be followed by a hug and a kiss. Thus she indicates her love for him as her child, but her disapproval of his behavior.

Finally, a few words about toilet training. Again, there are two ways of going about it. One is the reflex catching of stools and/or urine beginning at any age after the child is able to sit well unsupported. It should be noted that this is not training. True training is not possible until the child has matured sufficiently to understand the sensation of needing to empty his bladder or bowels, seeks a repository, and is capable of releasing urine or feces into it. Most children are eighteen months to two-and-a-half or even three years of age before this degree of maturity is reached. It is best to postpone training attempts until this older age period is reached.

When training is begun, the approach should be not to try to catch every urination on the hour or every stool at exactly one-half hour after breakfast, but rather to teach the child how people use toilet seats. It may seem to the adult that anyone ought to know enough to go to the toilet, but it must be remembered that infants do not; they have to learn the technique. Usually a nursery chair is preferable to a "toidy" seat on the adult stool. If the latter is used, a stepladder should be provided so the child can climb up by himself. The child should be directed to go to the toilet seat as near to the time as possible when a bowel movement or urination is to be expected. He should then be told to get his clothes off and as much assistance as necessary should be provided. He should get onto the toilet seat by simself and be left on for a period of a few minutes. Whether he performs or not, he is shown how to wipe himself and how to flush the toilet if the adult toilet is being used. He should be allowed to get off the toilet seat by himself and assisted in putting his clothes back on. Contrast this with the usual procedure when the mother carries the child to the toilet, lays him down on the floor or bed, takes off his pants herself, places him on the toilet, wipes him herself, lifts him off, puts his clothing back on, and then lets him go. Mother is beautifully trained, but the child has no responsibility whatever. Many children at two years of age can go to the toilet entirely by themselves. There is, however, no particular objection to mothers placing their infants on the toilet seat at an earlier age in an attempt to "catch" the bowel movements, if they wish to do so. Often this succeeds very well, but there should be no illusions about calling it training, for it is not. It is merely "catching" and should be abandoned at the first sign of objection on the part of the infant. The latter is equally true when objections are encountered during training attempts at the older age period. Conditioning against the toilet seat, which may delay training many months, is a real risk when attempts are made too frequently or when over-anxious or unpleasantness is exhibited by the mother.

The child may emerge at the end of the two-year period feeding himself, sleeping throughout the night, reasonably happy in his daytime play, not using temper-tantrums excessively as an attention-getting device, and perhaps with some degree of success in use of the toilet. If so, living together should be a pleasant experience for both child and parents. Furthermore, the basis for the continued development of healthy mental attitudes should have been soundly laid.

In the subsequent years of childhood there are other characteristic patterns of behavior which will not be discussed in detail here. Advance warning at the appropriate age period concerning these should be an assumed responsibility of the physician in his care of the well child, however. A few illustrations may be cited. Between two and three years of age most children show an interest in wanting to dress and undress themselves. Assisted, the task can be accomplished to a surprising degree by the age of three or a little after. Practically all children can be taught to take their own baths by two and a half to three years of age. Fears

manifest themselves between the ages of two and four, and vivid imagination crops up during the same period. "Sassiness" can be expected between four and six. Children try out expressions they have heard such as "you old dummy," "I hate you," and "Shut up." Ignored, the use of such language soon dies out. When reacted to violently, however, as commonly occurs among parents, children tend to continue its use because it has been found effective.

One final illustration should be mentioned because of its possible serious consequences. This is the occurrence of body curiosity in the opposite sex which may be expected to appear in many children around the age of five or six. Children of the opposite sex inspect one anothers' genitals out of curiosity to note how they differ. Such behavior is no indication that these children are on the road to becoming sex perverts in later years, but by the violent reactions of some parents it is obvious that they think so. Clearly, the sex idea is more in the adult mind than in the child's. Magnification of the incident out of all proportion to its significance runs the risk of leaving a lasting impression on the child's mind. Parents should be forewarned to meet the acute situation calmly but not to ignore it completely. What is called for is instruction in social customs which decree that we keep our clothes on in public but do as we please in the privacy of our own homes.

Summary

Within reasonable variations most normal children pass through patterns of behavior at different age periods which can be regarded as expected. Physicians can render a helpful service to parents by anticipating for them and interpreting to them these expected patterns of behavior. The objective is to secure more daily satisfaction for parents in living with their children and to prevent later more serious behavior problems.

Expected patterns of behavior selected for discussion in the first year of life include infant crying or "colic," thumb sucking, fear of strangers, and body curiosity.

The importance of the second year of life in the establishment of desirable behavior attitudes has been stressed. Eating, sleeping, play, tempertantrums, and toilet training have been considered in detail.

Additional expected behavior in later age periods has been mentioned briefly. Body curiosity in the opposite sex is a not infrequent form of normal behavior among five and six year olds. Its management to avoid lasting psychic trauma to the child has been emphasized.

RESPONSIBILITY FOR MATERNAL MORTALITY

(Continued from Page 110)

organizations and groups interested in the problem. We therefore welcome the suggestion by Drs. Freeman and Barno that a detailed workable set of recommendations for the organization, regulation and conduct of obstetrical departments in Minnesota hospitals be developed by representatives of state and local medical societies, state and local obstetrical societies and the state hospital association, in cooperation with the Minnesota Department of Health. We request the active cooperation and support of the Minnesota Obstetrical and Gynecological Society first, in developing and establishing effective recommendations for the proper hospital care of obstetrical patients; and second, and even more important, in stimulating hospital medical staffs to accept and carry out such recommendations. This approach should help reduce and ultimately to eliminate all possible preventable factors in maternal mortality.

FALSE ALBUMINURIA

(Continued from Page 111)

who are completing their treatment after discharge from a sanatorium by continuing their drug regimen even after returning to work, a false-positive test for albumin produced by the presence of PAS and certain preservatives in the urine is of sufficient importance to warrant the present report.

Bibliography

Bogen, Emil; Loomis, Russell N., and Will, Drake W.: Para-aminosalicylic acid treatment of tuberculosis—A review. Am. Rev. Tuberc., 61:226, 1957.

Lyght, Charles E.: Personal communication. (Merck, Sharp & Dohme Research Laboratories).

Way, E. Leong; Smith, Paul K.; Howie, Donald L.; Weiss, Rowena, and Swanson, Rollan: The absorption, distribution, excretion, and fate of para-aminosalicylic acid. J. Pharmacol. & Exper. Therap., 93:368, 1948.

Clinical-Pathological Conference

Sponsored by
The Minnesota Society
of Clinical Pathologists

CASE PRESENTATION—MINNEAPOLIS VA HOSPITAL

HENRY A. JOHNSEN, JR., M.D.
ALFRED EICHENHOLZ, M.D.
SAMUEL NESBITT, M.D., Ph.D.

This was the first and only admission of this patient to our hospital. He was a fifty-eight-year-old white married fuel oil salesman from St. Paul, who had complained of severe malaise with afternoon fevers and night sweats for three months. Just before the onset of these symptoms he had noted increasing fatigability and he decided to take his family with him on a vacation trip to Mexico. During the trip he developed a "cold" and after arrival experienced sweats and a fever as high as 102 degrees F. He was attended by a Mexican physician who made a diagnosis of pneumonia and gave him penicillin injections. While in Mexico the patient and members of his family experienced diarrhea. Soon after his return home he noted increasing weakness, anorexia, weight loss, afternoon temperature elevations and drenching night sweats.

Ten weeks before admission to the VA Hospital he entered a St. Paul hospital. At that time he appeared acutely ill and a definite hepatosplenomegaly was noted. No lymphadenopathy could be detected. Laboratory tests revealed a hemoglobin of 13 gm. per cent, a leukocyte count of 1500 (normal differential) and a sedimentation rate of 90 mm. in one hour. Agglutination tests for brucellosis, psittacosis, toxoplasmosis, tularemia and typhoid and paratyphoid fever were all normal. The serum Wassermann had converted to positive at that hospital two years previously, after many negative Wassermanns in previous years. It was again positive on this admission to the St. Paul hospital. Blood cultures and also tests for lupus erythematosis were negative as were coccidioidin, histoplasmin and Mantoux (1:100) skin tests. For the first time at that hospital serial chest x-rays revealed a large nodular lesion in the right upper lobe, which continued to enlarge during his hospitalization. Routine x-rays of the chest taken during previous admissions there for various surgical procedures were normal, including the most recent one taken just a few months previously.

Because of the positive Wassermann, he was given 15,000,000 units of penicillin, without any change in his condition. A course of chloromycetin was given for possible typhoid fever, without benefit. Chloroquin was given for a suspected malaria, although blood films for malaria organisms were negative. His condition continued to deteriorate. Biopsy of an inguinal node revealed only fibrosis. A bone marrow biopsy showed hyperplasia in the smear and granulomata in the sections. Staining of the granulomata by the Ziehl-Neelsen method revealed no acid-fast organisms. A consultant in internal medicine thought that lupus erythematosis should be considered and steroid therapy was subsequently recommended and given. The temperature returned briefly to normal but then rose again and the patient developed ankle edema. Because of the bone marrow granulomata Isoniazid, Streptomycin and PAS were added to the steroid therapy. The temperature again returned to normal and remained there but the sweats continued. He gained twelve pounds and was discharged from the hospital. He returned to work for a brief period of time but the severe sweats continued and within two weeks he was again running afternoon temperatures of 102 to 105 degrees. Three weeks after discharge he returned to the hospital, where it was noted that his hemoglobin was now 5 grams per cent and the leukocyte count was 900 cells per cubic millimeter. He was given four blood transfusions in the course of the next few days. He experienced chills almost daily and described approximately five episodes of severe rigor which shook the bed and which lasted from one-half to two hours each. Shortly before transfer to the VA Hospital he experienced an episode of squeezing tightness of the chest which was accompanied by dyspnea and which was relieved by oxygen.

The past history revealed that he had undergone gastric surgery in 1923, a tonsillectomy in 1934, removal of a kidney stone through a left lumbar approach and also a left inguinal herniorrhaphy in 1953, and a right inguinal herniorrhaphy a few months before admission. He had suffered an attack of "the flu" one year before the onset of his present symptoms. The family history was non-contributory and the system review was negative except for an episode of jaundice several years previously. He had received several prostatic massages at some time in the past.

Physical examination on admission to the VA Hospital disclosed a well-developed, sallow man with evidence of recent weight loss. He spoke in a tired, monotonous voice and appeared to be in considerable distress. He was 6 feet, 2 inches tall and weighed 173 pounds. His temperature was 102 degrees F., his pulse rate 84 and his blood pressure 108/58. The sclerae were icteric. Blood oozed almost continuously from both nostrils. The tongue was coated and the breath was fetid. He was edentulous and a copious post-nasal drip was present. An ulcer was present in the left tonsillar fossa, on the right side of the lower lip and on the chin. "Fever blisters" were also present on the lips. Hard, slightly tender, enlarged inguinal and cervical lymph nodes were palpated. Post-tussive râles were heard posteriorly over both lung bases. The vital capacity was 3.6 litres, rapidly expelled. Well-healed scars of previous surgical incisions were noted over the abdomen, the groins and the left flank. The liver edge was palpable 14 cm. below

Dr. Johnsen is Instructor in Medicine and Instructor in Neurology and Psychiatry, University of Minnesota and is Physician-in-Charge, Psychiatry Research Laboratory, VA Hospital.

Dr. Eichenholz is Instructor in Medicine, University of Minnesota and is Staff Physician, Pulmonary Disease Section of the Medical Services, VA Hospital.

Dr. Nesbitt was formerly Associate Professor of Medicine, University of Minnesota and Attending Physician, Medical Services, VA Hospital. Present address: 25 East 83rd Street, New York 28, N. Y.

Dr. Gleason is Chief of Pathology, VA Hospital.

Dr. Hagen is Associate Professor of Medicine, University of Minnesota and is Chief, Hematology Section of the Medical Services, VA Hospital.

Requests for reprints should be addressed to Henry A. Johnsen, Jr., M.D., Neuropsychiatry Service, VA Hospital, Minneapolis 17, Minnesota.

the right costal margin in the mid-clavicular line and it was firm and tender. The edge of the spleen was palpated 6 cm. below the left costal margin in the mid-clavicular line and it was firm and non-tender. A pronounced, pulsatile palmar erythema was seen. The left lobe of the prostate was firm, non-tender and nodular. Hemorrhoids and varicose veins of the legs were present and the ankles were slightly edematous. The Babinski was thought to be positive on the right by one observer. The patient was very weak and walked with a staggering gait. The examination was otherwise within normal limits.

Laboratory tests revealed that the hemoglobin ranged from 10.9 to 9.6 gms. per cent on five determinations, with red cell indices on the latter determination as follows: mean cell diameter 7.4 microns, mean cell volume 95 cubic microns, mean corpuscular hemoglobin 28.5 micro-micrograms and mean corpuscular hemoglobin concentration 30 per cent. The hematocrit was 32 per cent and the red cell count was 3,350,000 per cubic millimeter. The leukocyte count ranged from 1350 to 2350 on four determinations, with normal differential counts. Two platelet counts were 14,000 and 40,000 per cubic millimeter. Two reticulocyte counts were 4.6 and 6.5 per cent. Direct and indirect Coombs' tests were negative. The erythrocyte sedimentation rate was successively 75, 83 and 80 millimeters in one hour, Westergren method. The bleeding time was seven minutes and the coagulation time was twenty-one minutes. A blood film for malaria organisms and two clot tests for lupus erythematosis were negative.

Two urinalyses revealed specific gravities of 1.017 and 1.023, albumin 2 plus and 4 plus and the tests for reducing agents were negative. The more concentrated specimen tested negative for bile, but contained 5 to 10 leukocytes per high powered field, 3 to 5 red blood cells per high powered field and 20 to 30 coarse granular casts per high powered field.

Liver function studies revealed that the serum bilirubin was 1.0 mgm. per cent on the one minute direct test. The total serum bilirubin was 2.6 mgm. per cent. The twenty-four-hour urinary urobilinogen excretion was 8.15 mgm. The cephalin flocculation was 4 plus in twenty-four and forty-eight hours. The thymol turbidity was 8.5 units. The zinc sulfate turbidity was 13.8 units. The prothrombin activity was 47 per cent. The total serum cholesterol was 105 mgm. per cent. The bromsulphalein retention was 27.1 per cent in forty-five minutes, when the serum bilirubin was as reported above and when the oral temperature was 101.6 degrees F. The total serum proteins were 5.9 grams per cent. These were subjected to electrophoretic separation with results as follows: albumin 2.5 grams per cent or 42.30 per cent, $alpha_1$ globulin .2 grams per cent or 3.55 per cent, $alpha_2$ globulin .5 grams per cent or 8 30 per cent, beta globulin .5 grams per cent or 8.30 per cent and gamma globulin 2.2 grams per cent or 37.55 per cent.

Evaluation of blood chemistries revealed that the blood urea nitrogen was 24.5 mgm. per cent on admission. It rose in seven days to 65.0 mgm. per cent on the day before death. The plasma CO_2 combining power on the same days was 30.2 and 7.5 meq./l. On admission the serum chlorides were 92.7 meq./l., the serum sodium was 130.4 meq./l. and the serum potassium was 4.5 meq./l. The serum alkaline phosphatase was 10.0 KAU and the acid phosphatase was 2.8 KAU. The admission fasting blood sugar was 106 mgm. per cent (99-100 mgm. per cent is the upper limit of normal in our laboratory.)

In a search for an infectious disease, the bronchial washings were smeared and cultured for acid-fast bacilli, but none were found. Blood cultured the day after admission grew out a non-hemolytic streptococcus in broth only. Another blood culture the next day was negative. Culture of a mid-stream urine sample grew out 3 000 colonies per milliliter of hemolytic and non-hemolytic staphylococci which were coagulase negative. Three purged stools were negative for parasites or their ova. Two routine stools were negative for blood. Cold agglutinins and serum agglutinins for brucella antigens and for typhoid H antigen were not present. Serum agglutinins for typhoid O and paratyphoid A & B antigens were all present in a dilution of 1:20. Heterophile agglutinins were present in a dilution of 1:14. A test for serum C-reactive protein was 3 plus. The VDRL slide test was positive in a dilution of 1:32 and the Wassermann complement fixation was positive. A test for serum cryoglobulins was negative. The treponemal immobilization test was positive.

An electrocardiogram was normal. X-rays of the chest, including planigraphy and bronchography, revealed a large homogeneous, lobulated mass in the right upper lung field in good focus from 9 to 14 cm. from the posterior thoracic wall. A mass in the right hilum was also noted. All the branches of the right bronchial tree could be filled with dye. One branch in the region of the mass was extremely irregular and narrow. Some cylindrical dilatation of the bronchi to the right upper lobe was present. The chest films of the patient's wife, son and daughter were reviewed and found to be normal.

The patient's hospital course was marked by fever with daily fluctuations between 99 and 102 degrees (oral). He was given prednisone daily during his evaluation in order to avoid sudden termination of the steroid therapy he had received elsewhere before admission. While being continued on steroid therapy, blastomycin, coccidioidin, histoplasmin and first strength PPD intradermal tests were administered however, and were all negative. Bronchoscopy was normal except for the presence of a non-ulcerated, smooth 5 mm. papule in the mucosa of the left main bronchus, the middle lobe orifice and the bronchus of the right medial basal segment. Microscopic examination of the washings revealed cells that led the pathologist to suspect a possible malignancy.

The tissue slide from the inguinal node biopsy performed at another hospital was sent for and reviewed and was interpreted as normal except for moderate fibrosis. Accordingly, on the fifth hospital day a biopsy of a left posterior cervical node was performed. By this time the patient's condition was deteriorating rapidly. His jaundice and edema had increased and he was troubled with anorexia, abdominal cramps, vomiting and diarrhea. He continued to perspire profusely, became dyspneic and apprehensive and on the eighth hospital day was obviously confused. His nose continued to ooze with a trickle of thin blood. His abdomen had become distended and very hard. Oxygen was administered but he complained of a smothering sensation with the mask on. On the ninth hospital day he was clammy and pale. His respirations became rapid and shallow and his pulse weaker and he finally expired before the cervical node biopsy report was received.

Discussion

DR. PAUL S. HAGEN: I shall assume that everyone present has read this protocol. Approximately one month after this man's illness began, it was noted that he had hepatosplenomegaly and leukopenia. In another hospital a biopsy of an inguinal node showed fibrosis. There is no indication that a touch preparation of the node was examined before routine sections were made. Apparently the node was not cultured. A bone marrow biopsy showed hyperplasia and numerous granulomata. A Ziehl-Neelsen stain showed no acid-fast organisms. There is nothing in the protocol to indicate that the marrow was cultured on specific media for various organisms.

DR. W. H. HALL: I don't believe the marrow was cultured.

DR. HAGEN: A chest film showed a nodular lesion in the right upper lobe. Before this conference began, I was told that a comparison of our own x-rays with those

of the referring St. Paul hospital revealed an increase in the size of the area of the infiltrate. He was tried on various courses of antibiotics and yet had severe shaking chills. In spite of the antibiotic therapy, there was a progression of the pulmonary lesion.

A series of laboratory studies are described. From a hematologist's viewpoint, the essential points are that he had an anemia and a leukopenia. This was not a neutropenia, but a leukopenia. The reticulocyte counts were elevated and the platelet counts were low. The bleeding time was increased, compatible with the low platelet count. The anemia was normochromic and normocytic. I would like to defer further discussion of the laboratory test results until I have had an opportunity to see the patient's x-rays.

ROENTGENOLOGY RESIDENT: All the available films were taken within a few days of one another. Chest films disclose a rather round lesion which is fairly well demarcated from the surrounding lung (Fig. 1a). Some radiolucency within the lesion suggests cavitation. On the lateral projection the mass overlies the hilar shadows and for that reason we cannot make out its limits well (Fig. 1b). In addition, on the PA film we see hilar enlargement. Some scoliosis tends to accentuate the hilum. On planigraphy of the right upper lung field, one can see the minor fissure limiting the lesion inferiorly, so we know that this lesion is in the upper lobe. There is a lobulated appearance to the enlarged hilum, suggesting a hilar adenopathy (Fig. 1c). It does not appear to be present on the opposite side. The major branches of the bronchial tree are seen on several cuts and they appear patent and in their normal relationships. On bronchography there is fairly good filling of the bronchial tree. The lower and middle lobe bronchi appear normal. The upper lobe bronchi are well seen on the lateral film and show some changes suggestive of chronic bronchitis. There is slight irregularity to the bronchial wall. There are small filling defects suggestive of excessive secretion in the bronchial tree and the bronchi do not taper in the normal fashion. We can identify no bronchial connection between this lesion and the bronchi demonstrated. However, the mass lies adjacent to the pleura. It is not infrequent for one to fail to demonstrate a connection with a lesion in that position.

DR. HAGEN: Could you identify calcium within the subpleural or hilar masses?

RADIOLOGIST: No.

DR. HAGEN: Obviously we need further help from the laboratory in order to establish a diagnosis here. I want to point out once again that we are faced with a chronic fever unresponsive to antibiotics, including antituberculous agents. One needs to know whether antituberculous drugs were given for an adequate length of time to conclude that there was no response. Were they given for at least two months?

DR. NESBITT: I am sure they were not.

DR. HAGEN: Were they given for more than two weeks?

DR. NESBITT: That was about the total length of his stay in that hospital.

DR. HAGEN: The serum protein values may be of some help. On electrophoresis the albumin was moderately decreased and the gamma globulin markedly increased. In tuberculosis an increase in alpha globulin is more likely. The BUN was elevated. It is very interesting that certain agglutinations either were not obtained or were purposely not described. With this type of clinical situation there were certainly some other agglutinations that I think the physicians on the ward must have obtained.

A hemolytic anemia was possible because of the elevated reticulocyte counts. The thymol and zinc sulfate turbidities were elevated. These suggest that an immunochemical reaction could be responsible for the anemia and leukopenia. This is a possible explanation of the peripheral pancytopenia, because the bone marrow done at the other hospital was found to be hyperplastic. So the anemia and leukopenia were not due to depression of bone marrow activity but were more apt to be due to a hyperimmune mechanism. The protocol states that one blood culture was negative and one grew a non-hemolytic streptococcus in broth only. I wonder if we know how long these blood cultures were held.

DR. HALL: They were cultured for 5 days.

DR. HAGEN: The blood cultures may have been thrown away too soon for other than routine pathogens. I don't see any mention here of the inoculation of laboratory animals and perhaps it is not fair to ask, because this may not have been done or the results may not have been reported by the time the patient died. I presume the urine was also cultured for just a short time.

DR. HALL: Forty-eight hours.

DR. HAGEN: We may have missed an organism that is not one of the usual pathogens by not suspecting its presence before death and therefore not alerting the laboratory to the need for special culture techniques. The negative skin tests do not mean much in the presence of long term steroid therapy or in a patient that is overwhelmed by one of the organisms one is testing for. These tests are based on hypersensitivity, which may be lacking in such a patient. The patient might have been in an anergic phase of reaction to invasion.

How can we put this together? A possible explanation is one I am sure I am supposed to trip over, namely malignant lymphoma. One could postulate that he developed a lymphoma two years before he came in with a high fever and that the lymphoma produced an immune reaction which turned his serologic test for syphilis positive. Not only could the serologic test for syphilis change two years before a patient presented with fever due to lymphoma but also a positive Coombs' test could have been present the same length of time. We have recently had a patient that we had carried as an idiopathic acquired hemolytic anemia for almost two years. This was subsequently found to be due to Hodgkin's disease. Hepatosplenomegaly, leukopenia, probable hemolytic anemia and a hyperimmune process are perfectly compatible with a lymphoma. The serum protein electrophoretic curve with an elevated gamma globulin is also compatible with a lymphoma, although the ones we have seen have not had a marked elevation. The marked elevation of the gamma globulin was more consistent with an infection than with a lymphoma. There is a physical finding here that I think is of some importance. The liver was not only enlarged but was also tender, whereas the spleen was not tender. I have been trying to remember any patient that I have seen with a lymphoma who had a definitely tender liver. I wonder how tender this liver was. I think it is unusual to have a tender liver because of a lymphoma and I intend to take that as another sign that I had better get away from that diagnosis. Furthermore whenever one has a patient whose course is continuously febrile and whose fever does not relent with any antibiotic, a mycosis, with or without a lymphoma should be considered. I think that we have to give that diagnosis serious consideration in this patient. We should remember that it is not uncommon for patients with a lymphoma to develop an infectious disease and it is quite possible that this was the sequence of events with this patient. However it does not seem necessary to include the diagnosis of lymphoma when the entire picture can be explained by a certain infectious disease alone.

Disseminated tuberculosis is a possibility and is difficult to rule out. The failure of the patient to respond to antituberculous chemotherapy does not exclude this disease because he did not receive this treatment long enough for an adequate trial of therapy. He did indeed improve enough to be discharged from the referring hospital but his symptoms never left him completely and he had to return very shortly thereafter for his final admission there, because of a severe exacerbation of his illness. The negative skin test does not exclude tuberculosis for reasons already discussed. Acid fast organisms could not be found in his bone marrow granulomata or in his bronchial washings. While one would not expect to find the bacilli in his bronchial washings after miliary dissemination from a primary focus, one would expect to find them in this case because his pulmonary lesion is a large mass which is probably cavitary. A bronchial communication with the lesion would have to be present however. For these reasons, disseminated tuberculosis would not be my first choice.

A bronchogenic carcinoma with widespread metastases is a possibility but an unlikely one. The roentgenologist suspected cavitation of the pulmonary mass which, it is true, could be central degeneration of a tumor. However an infectious etiology is more likely. Suspicious cells were noted in the bronchial washings, but, again, these could just as likely be a proliferative response to infection. No occlusion of a bronchus can be seen on the planigrams and no "rat-tail" deformity characteristic of malignancy is seen on the bronchograms. If calcium were present in the subpleural or hilar masses, I would feel more secure in saying this was an infectious granulomatous process rather than a malignancy. On the other hand, granulomata were seen in the bone marrow and presumably no tumor cells were seen there. If the pulmonary lesions are also granulomatous they may not have been present long enough for calcium to form.

The ulcer in the tonsillar fossa in combination with the pulmonary lesion, hepatosplenomegaly, leukopenia, anemia, chronic unremitting fever in spite of antibiotics, night sweats, emaciation, and death within several weeks after the onset of symptoms are all highly suggestive of disseminated histoplasmosis. This man's residence in the Mississippi Valley is also consistent with that diagnosis and disseminated histoplasmosis is the diagnosis I am going to hang my hat on in this conference. The way to prove the diagnosis is to observe or grow the organism. This would not be likely with routine culture techniques and that is the reason I have been so inquisitive about the techniques used. One serologic test that is not mentioned in the protocol is the complement fixation test for histoplasmosis. For this test, we send our specimens to the Bacteriology Department of the Army Medical Service Graduate School at Walter Reed Army Medical Center in Washington, D. C. If this was done, there would not have been enough time for the result to have been returned to Minnesota before the patient expired. Therefore, since it would not have been available to the ward physicians before death, such a test may not have been included in the protocol.

DR. NESBITT: We felt that we were very fair with Dr. Hagen. We gave him all the information we had up to the time of death. Shortly thereafter additional facts became available. The first of these was the report on the cervical node biopsy.

May we have the diagnoses offered by the medical students?

DR. E. B. FLINK: The following diagnoses were suggested: coccidiomycosis, tuberculosis, sarcoidosis, amoebiasis, histoplasmosis, paroxysmal hemoglobinuria and Hodgkin's disease.

Dr. Hagen's Diagnosis

Disseminated histoplasmosis.

Pathologic Findings

DR. D. F. GLEASON: I think we have been treated to a fascinating demonstration of CPCmanship in this very beautiful case discussion. The permanent sections of the lymph node biopsy became available the night before the patient died. Histoplasma capsulatum was identified on the slides. Permission for an autopsy was initially refused by the patient's wife. However when the lymph node picture indicated that this case was an example of disseminated histoplasmosis, the wife was contacted again and was finally persuaded to grant an autopsy out at the mortuary on the day after the body was embalmed. Permission was limited to examination of the trunk only, however. The embalming process prevented us from obtaining accurate weights of the various organs and their appearances also were altered.

Upon gross examination an abscess was noted in the anterior segment of the right upper lobe. The abscess measured 4½ centimeters in diameter and had a liquid center, surrounded by a soft wall of connective tissue. Presumably this was the large round lesion seen on the x-rays. Numerous smaller soft nodular lesions were seen throughout the lung and another abscess, which measured one centimeter in diameter, was found on the other side of the chest in the left lower lobe. The liver was very large. Its weight was estimated at 4500 grams. The spleen was also very large, apparently weighing about 2000 grams. What appeared to be Malphighian corpuscles on first glance were actually small yellow abscesses or infiltrates. They were somewhat larger than Malphighian corpuscles, measuring about four millimeters in diameter. The kidneys also had very tiny yellow nodules scattered through them. The periaortic and hilar lymph nodes were enlarged and were quite soft and mushy. An incidental finding of chronic cholecystitis was also noted. The remainder of the organs within the trunk appeared grossly normal.

Microscopic examination of non-caseous material from the lung, stained with hemotoxylin and eosin, reveals myriads of macrophages, and even capillary endothelial cells, which contain intracellular organisms with a central body that stains with hemotoxylin and a clear capsule that does not take any stain. This Giemsa stain of the same section makes the capsule a little more visible. With this periodic acid-Schiff stain it can be seen that these intracellular organisms are Schiff positive. The central body stains a dark red and the capsule is also Schiff positive, staining a lighter shade of red. Similar intracellular Schiff positive organisms can be seen in the spleen (Fig. 1E), in the endothelial cells of the capillaries of the renal glomerulus (Fig. 1F), in these miliary nodules in the liver (Fig. 1G), in almost any lymph node (Fig. 1H) and in the bone marrow. The bone marrow is very extensively involved, even being necrotic in some areas. We wrote to the referring hospital for the bone marrow biopsy that reportedly contained granulomata, hoping to be able to identify the organism. We never received that marrow for review, however.

DR. HAGEN: The organisms you showed us in a lymph node were from a section, weren't they?

DR. GLEASON: Yes, these are all sections. We made some touch preparations from the lymph node and the organisms were seen by that method also.

Try to remember the relative size of the organisms you have just seen. We will now look at some sections from the center of that lung abscess. It is filled with necrotic debris. Under the high dry power and with routine hemotoxylin and eosin staining we find it difficult to identify a causative organism. Many similar

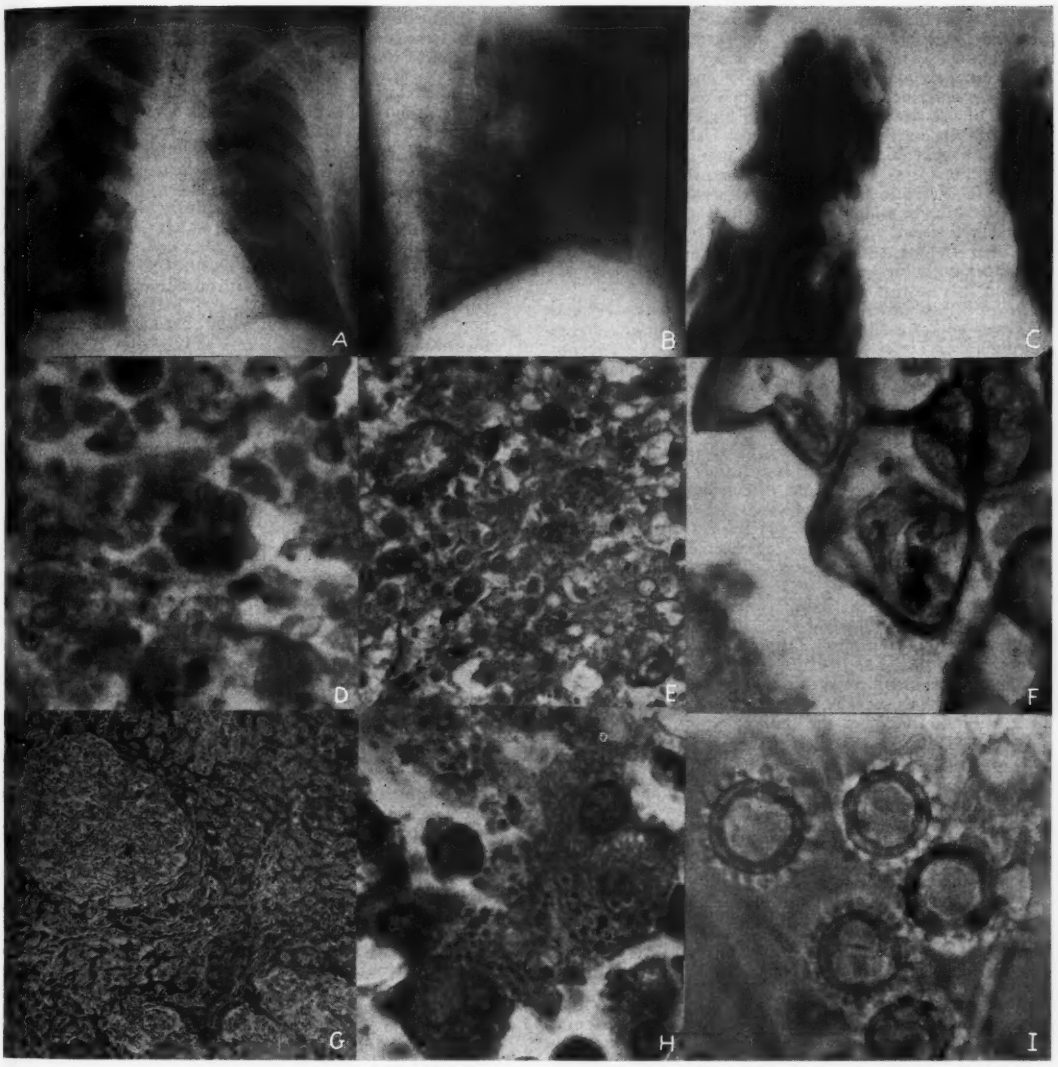


Fig. 1. Disseminated histoplasmosis. (A) Right subpleural and hilar masses on routine PA film of chest. (B) Lateral view with mass overlying hilar shadow. (C) Planigram showing lobulation of hilar mass and no visualization of calcium in either mass. (D) Puckett bodies in subpleural necrotic mass. (E) Intracellular H. capsulatum is seen in macrophages in the spleen and (F) in endothelial cells of a glomerular capillary. (G) Miliary nodules in the liver with numerous organisms seen. (H) Intracellular organisms in lymph node macrophages. (I) Tuberculate chlamydospores of H. capsulatum obtained on culture of a hilar node.

Photography by Medical Illustration Service, VA Hospital, Minneapolis. Selection of subject matter and supervision of photography by Martin A. Segal, M.D., formerly Assistant Pathologist, VA Hospital, and now Pathologist, Asbury Methodist Hospital, Minneapolis, Minnesota.

but smaller pulmonary nodules have been resected from other patients with similar findings on pathologic examination. In some instances they have been called "tuberculomas" or "idiopathic pulmonary granulomata, probably tuberculous." These "coin" lesions are usually rounded subpleural nodules with a fibrous capsule and a laminated necrotic center. Routine stains show no organisms and Ziehl-Neelsen stains show no tubercule bacilli. Puckett[1] has shown that many of these lesions, when stained with periodic acid-Schiff stain and examined under oil immersion, contain numerous encapsulated organisms which are now extracellular but which bear a close resemblance to Histoplasma capsulatum. The only objection to a diagnosis of histoplasmosis has been that the organisms will not grow in culture media.

Examination under oil immersion of the necrotic material from one of this patient's lung abscesses, which has been stained with periodic acid-Schiff stain, now reveals extracellular histoplasma organisms (Fig. 1D). However, many of them are almost twice the size of the intracellular organisms I have just shown you. Dr. Segal actually measured them and noted a statistical difference in the average sizes of the organisms in the center of these areas of necrosis. The central body in

these large Puckett organisms is more hyperchromatic and the space between it and the capsular envelope is quite empty. The patient we are presenting in more detail today is the same one whose histologic findings only were described by Dr. Segal[2] before the Midwestern Section of the American Federation for Clinical Research at one of its meetings in Chicago. Using this patient, Dr. Segal was able to bridge the gap between typical intracellular forms of Histoplasma capsulatum and the bodies in necrotic debris which were described by Puckett. All variations of these forms, from the classic intracellular forms to those described by Puckett, can be identified in the sections from this one proven case. The Puckett bodies do not stain with hematoxylin and eosin in this abscess.

One of this patient's lymph nodes was cultured and it grew out organisms with a characteristic colonial morphology. These are "chlamydospores," which means spores with a mantle or protective envelope. The envelope is studded with periform tubercles (Fig. 11). Tuberculate chlamydospores of this size and appearance are the proof positive of histoplasma in culture.

DR. NESBITT: Dr. Hagen is certainly to be congratulated for his very excellent discussion. Other things which he asked for and which did eventually become available after the death of the patient were the serological tests for fungi. The complement fixation test for coccidioides was negative. The complement fixation test for blastomyces was positive in a dilution of 1:32. The complement fixation test for histoplasma was also positive in a dilution of 1:32. The collodion agglutination test for histoplasmosis was positive in a dilution of 1:8.

The bronchoscopic washings also grew out Histoplasma capsulatum, the report being received some time after the death of the patient.

DR. JOHNSEN: It is interesting to note that the first case of disseminated histoplasmosis ever described in the United States was that of a Minnesota woman. I believe this case was reported by Dr. C. J. Watson[3] of the University of Minnesota, who did the autopsy. The disease was first reported by Darling as a kala-azar type of syndrome in Panama. Dr. Watson[4] later described the pathology of the disease in detail.

DR. HALL: The complement fixation test was done at Walter Reed Army Medical Center. We have frequently received reports of cross reactions. Complement fixation tests positive in titers of 1:8 or 1:16 for blastomyces in cases that we think otherwise are histoplasmosis by skin test and by clinical features have been frequent. This present patient is the only case that we have had that has been proven by culture to be histoplasmosis. Most of our other cases have been those with a solitary granuloma. These contain the large extracellular organisms and they have failed to grow in culture.

The positive TPI test is a surprise because this patient was questioned about syphilis several times and he was said to have denied any previous lesions or any treatment for syphilis. Many people regard the TPI test as being absolutely specific for syphilis. It is, however, a test which remains positive even though the disease becomes inactive or is treated successfully. I don't know how to explain the two-year history of a positive Wasserman and a positive TPI test.

DR. JOHNSEN: We don't know how long this man had been host to the histoplasma organism. I wonder if it is not possible that he was harboring the organism two years ago and that the conversion of his VDRL, Wassermann and TPI tests was another example of a cross-reaction to histoplasmosis. Perhaps Histoplasma capsulatum contains antigens that are not only chemically similar to those of Coccidioides immitis and Blastomyces dermatitidis (which we know to be true because of skin tests and serologic tests) but are also chemically similar to those of Treponema pallidum.

DR. HALL: I think there might have been some value in examining his blood film more closely. We might have seen the organisms in his peripheral blood. No organisms were found in the lymph node from the other hospital but I suppose they could have been there and could have been missed. The only tissue from which we got a positive culture was a hilar lymph node. I would also like to say that if a sizable piece of necrotic material can be obtained, even though it has been in formalin, it is worthwhile to wash it and to cut into the center of it to obtain material for a culture. This case proves that all of the organisms may not be killed by the formalin, because of incomplete saturation of the tissue.

DR. R. E. SMITH: What was the working diagnosis before death?

DR. NESBITT: The diagnoses considered by the clinicians on the ward were malignant lymphoma, bronchogenic carcinoma with metastases and a possible Budd-Chiari syndrome. Dr. Horace Zinneman held out for histoplasmosis.

DR. SMITH: I would like to hear Dr. Hall's comments on therapy.

DR. HALL: There isn't any therapy.

MEDICAL RESIDENT: Does anyone feel that the organism became more disseminated because of steroid therapy?

DR. HALL: Well, that is possible. Certainly, the organisms were very numerous, being found even in the endothelial cells of the capillaries. Not having effective chemotherapy, I think it is reasonable to think that the steroids might have caused further dissemination. However, one can see a similar course without steroids. He already had the symptoms and signs of dissemination by the time he was admitted to the referring hospital, which was before steroids were given there.

DR. ZINNEMAN: I would like to ask Dr. Gleason what the adrenal glands looked like.

DR. GLEASON: A few macrophages in the sinusoids of the adrenal cortex contained the organisms but there wasn't any necrosis or focus of inflammation.

DR. HALL: Many cases of disseminated histoplasmosis die with Addison's Syndrome because of destruction of both adrenal cortices.

DR. HAGEN: The relatively low blood pressure and low serum sodium made me wonder about a secondary adrenal insufficiency here.

DR. NESBITT: Dr. Flink is interested in the impression of the medical student who really did a very fine job in working up this case. He included histoplasmosis in his differential diagnosis.

Final Anatomical Diagnosis

Disseminated histoplasmosis.

References

1. Puckett, T. F.: Am. Rev. Tuberc., 67:453, 1953.
2. Segal, M. A.: Clinical Research Proc., 3:208, 1955.
3. Watson, C. J., and Reilly, W. A.: Arch. Path. & Lab. Med., 1:662, 1926.
4. Watson, C. J.: Folia haemat., 37:70, 1928.

Editorials

JOHN F. BRIGGS, M.D.
ARTHUR H. WELLS, M.D.
HENRY G. MOEHRING, M.D.

TRENDS IN MEDICINE

It is trite but nonetheless true to say that we are living in a rapidly changing era. Medicine—both in health and in disease—has changed more rapidly in the past two decades than in all of its history, and the end is not in sight. So rapid and profound are these changes that those of us who are living through them, like passengers in an express train, have only a kaleidoscopic picture of our changing surroundings. It is my purpose in these comments to discuss the present status of medical care, to outline some of our current problems, and to enthuse about a new and idealistic program for the prevention of disease.

As far as physical illness is concerned, the advent of chemotherapy has virtually eliminated most infectious diseases and will probably soon reduce the few remaining to unimportance. Malignant illness is not immune to our attack and may soon succumb. Even degenerative illness shows promise of conquest. Surgical advances grow ever bolder and more imaginative. I believe we shall even see a completely synthetic heart—doubtless of plastic—replacing a damaged one, activated by a pocket battery with a rheostat to permit greater emotional or physical exertion! Scarcely an organ in the body has not been proudly removed and often replaced by a graft of some type, and all sorts of trauma yield to the skill of the surgeon—internist — anesthetist — physiologist — et al who comprise the modern therapeutic team. In the exciting drama of modern medicine, we bemoan the general practitioner and write diatribes against the age of specialization—saying much the same that was said forty years ago or longer. We have coined a new word "generalist," though what is encompassed in the practice of this modern superman is not defined. Intelligently we regard the family doctor, usually an internist, as a conductor of an orchestra. It is he who controls the medical destiny of his patient, but it is he who calls upon the skills of others when his own are inadequate. This would appear to be the acme of complete medical care, not only for the individual, but also for the family—for we know that no man is an island but an archipelago with parents, spouse, children, friends and associates, boss and subordinates influencing every symptom which he has.

The field of emotional illness has expanded also. Not only do we conquer or control intractable insanity with weirdly formulated chemicals for which we have coined jaw-breaking names, but we also feel ourselves to be pioneers in discussing "psychosomatic illness," which is in fact as old as man himself. I believe Paul White has described the mind and body as bearing the same relationship as an electric current and a magnetic field—neither can be altered without affecting the other. Scarcely an illness or an accident lacks its emotional response; scarcely an emotional storm or disappointment, irritation or ambition, fails to have some associated physical reaction, be it diarrhea, pain, headache, sweat or palpitation. Let us not, therefore, speak of mind and body as separate but as co-ordinated apparatuses. They are in reality inseparable as salt and water in the ocean.

Equally important, though less susceptible to observation and analysis, is the spirit. No physician worthy of the name but respects the power of courage to heal, and of fear to destroy. No physician has cared for the sick without bowing his head to the power of prayer and faith to overcome impossible obstacles. Any thinking person respects and acknowledges the healing and refreshing power of meditation, solitude, faith and prayer.

It may seem strange to bring faith, ambition, and specialization together with antibiotics, surgery, and the family doctor. Yet this is the essence of care of the whole man. We cannot, as practitioners of the healing art, content ourselves with prescribing pills or injections, with amputating or replacing, or with auditing the recollections of the conscious and the sub-conscious. It is properly our function to investigate and to understand family relationships, economic and social pressures, and the religious faith of those who entrust their health—in its broadest sense—to our care and to formulate from this, concrete and practicable recommendations.

First of three editorials on Trends in Medicine.

The periodic health examination when properly done is a tremendous advance in medicine. By this examination, the physician may anticipate trouble, and by anticipating may prevent. But the scope of such an examination is awesome. Not only must the physician take a complete history, not only must he do a thoughtful physical examination, but he must also gain by question and intuition some knowledge of the emotional life, the social career, the economic problems, the ambitions, *vis-a-vis* the accomplishments of his patient. Much of this knowledge comes by osmosis during the examination. In smaller communities, the doctor's social contact with the patient fills other gaps. Often the more pressing problems are volunteered, provided the physician is receptive and appreciative. But there is always a residue which must be deliberately sought if we are to do the best job. The money fears, the shame over past errors, the feelings of inadequacy, the problems of sexual relationships, are seldom offered unless questioned. Last but not least are the indispensable laboratory tests and x-rays. The latter raise the dilemma of irradiation hazard in the physician's mind.

Can such a complete health examination ever be actually accomplished? I suspect the answer is no, but completeness can be approached if we bear in mind the complexity of the problem, and if we are as sensitive to things unsaid as to those spoken. But we must always realize that the best preventive medicine should not end with cancer prevention, detection of early diabetes or hypertension, or with the recognition of existing illness in an early stage. The best in preventive medicine means a positive approach to good health, an approach which goes beyond illness, which suggests and implements things which can and should be done to attain maximum health, optimum pleasure and productivity. This approach must include a scientific medical examination, a delicate and perceptive emotional evaluation, and spiritual or intellectual feelers equally delicate and equally important. Only through this approach to the whole man can the physician discharge his treasured responsibilities. His final step, however, must be to offer positive recommendations rather than a statement of negative findings.

For this reason, the family physician holds the highest position in medicine which a man can have. But this is also why medical care is ever changing and ever challenging. This is why we must be ever alert to growth. This is why one speaks of "practice" of medicine.

CHARLES S. HOUSTON, M.D.
Aspen, Colorado

GOVERNMENT

Government exists for the people. And the nature of the population has a major effect on the problems of government.

One of the most important factors in our rapidly changing society today is the growth in our population. In any planning for the immediate future we must take into consideration, not only the increased numbers, but the changing composition of this total population. Sensible decisions cannot be made unless the following facts are considered.

In 1946, Minnesota's total population was 2,734,000. In 1955, it was 3,174,000. In 1966, it is expected to be 3,600,000. If we keep government services only at the present level, it is obvious that the volume of services will have to be increased to keep pace with our increasing numbers. And if—as has been true in the past—the people want more and better services, such as more years of education for more of our children, the services will have to be increased even more.

Another important fact about our population is that it is changing in nature as well as in total numbers. The two age groups that show the greatest increase are those under twenty-one years of age and those over sixty-five.

One calculation of changes during the fifteen-year period between 1950 and 1965 shows that, while our over-all population would increase 22 per cent, those under twenty-one years would increase 38 per cent, and those over sixty-five years would increase 35 per cent. In this same period, the number of people in the most productive years between twenty-one and sixty-five would increase only 9 per cent. And we all know that it is the young and the old that make the greatest demand on government services. We must plan to meet that demand.

The increased proportion of children and youth makes it imperative that we expand our educational facilities and do everything possible to secure enough well-qualified teachers. We need to build more schools and classrooms.

Another trend that we all welcome is that an increasing proportion of our young people are

Third in a series of editorials on Government.

seeking higher education. We hope and expect that this trend will continue, but it presents our colleges and university with the very real problem of financing rapid expansion. Expenditures for education are, in my opinion, the wisest investment we can make. But we must honestly face the problem of raising the money needed to make that investment.

The increasing proportion of older people likewise creates a financial problem, primarily because of the increasing cost of providing medical care for the recipients of Old Age Assistance.

In spite of the increase in the total number of citizens over sixty-five years of age, the number who receive Old Age Assistance is steadily declining. In 1946, 54,000 were receiving OAA; in 1956 this number was 51,500; and we estimate that about 45,000 will be receiving assistance in 1966. This decline is due to the progressively increasing number of people receiving old age and survivors' insurance benefits under the provisions of the Social Security Act. Thus our expenditures for maintenance under the OAA program do not create a serious problem.

But the continuing and increasing need of our older group of citizens for more medical care has made this program increasingly expensive since 1945, when unlimited medical care for Old Age Assistance recipients was authorized from state and county funds.

In 1946, only about 17 per cent of OAA recipients were medical cases. Today, 47 per cent receive medical care, and it is apparent this trend will continue. During the past ten years the cost of medical payments has risen from $1,500,000 in 1946 to $16,000,000 in 1956, and an expected $17,000,000 in 1957.

Over 70 per cent of these medical payments represent nursing home and hospital care costs. Today, about 4,500 nursing home beds are occupied by OAA recipients. About 1,000 old people who are now patients in mental hospitals could be cared for in nursing homes if they were available. We estimate that in 1966, 9,000 nursing home beds will be needed for OAA recipients alone.

We are gratified at the increased life span of our people. We are determined that all of our older citizens shall get the medical care they need. But we must honestly face the problem of increasing costs, and find some way to meet them.

ORVILLE FREEMAN
Governor of Minnesota

EMPHASIS ON DIETARY SOURCES OF THIAMINE

Concern for the trend toward low levels of thiamine in the American diet is being expressed with increased frequency. USDA nutritionists admit that the amounts of thiamine recommended by the Food and Nutrition Board of the National Research Council are difficult to achieve without careful planning.

Dr. Faith Clark took note of this when she addressed the Nutrition Education Conference in Washington in April, 1957. She said:

". . . Thiamine levels are little related to family income. Sources of thiamine in diets are chiefly grain products, meat (especially lean pork) and milk. Although consumption of milk and meat increases with income, that of grain products and of lean pork cuts generally decreases.

". . . Various nutrients are affected differently by changes in the economic situation. For example, a nutrition education program for a period of prosperity would not need to emphasize protein or niacin. When people in this country have more money to spend for food, they are likely to buy meat. *Thiamine,* on the other hand, *would need to be emphasized in a nutrition program for a prospering economy.* The high income diet is likely to mean a shift away from grain products and pork and the foods chosen to replace them are not equally good sources of thiamine."

This change in dietary pattern is not entirely due to our present prosperity. Dr. Herbert Pollack, in addressing the New York State Dietetic Association meeting in April, 1957, reported an increase in the incidence of thiamine deficiency and beri-beri among diabetics who had been restricted in their use of bread. This development, as a result of dietary restriction, strikes a sharp note of caution to members of the medical profession: When making dietary recommendations, due consideration should be given to the inclusion of substitute sources of thiamine.

Says Dr. Russell M. Wilder, world famous authority on vitamin deficiency disease:

"I worry most about deficiency of thiamine, because some time ago I was engaged in a very extensive study of the clinical effects of diets low in thiamine and have experimental proof that deficiency of this vitamin, a degree of deficiency so mild as to defy detection by the doctor, may provoke disturbances that are tremendously significant. Courage and emotional stability, with other qualities of character, deteriorate in thiamine deficiency before the nerves to the arms and legs are in any way affected; that is before neuritis has developed on which the doctor depends for a diagnosis."

The present emphasis on obesity has also caused many people to eliminate bread from the diet or restrict its use because of the erroneous concept that bread is a fattening food. This trend contributes to the dietary changes reported by Dr. Clark. Actually, bread contributes a higher proportion of one's needs for protein, thiamine, niacin, calcium, and iron than it does of one's need for calories. Because of this, bread serves a useful purpose in the reducing diet.

As a means of assuring a more adequate distribution of thiamine, riboflavin, niacin, and iron, the enrichment program was started in 1941. Dietary surveys had indicated that many diets did not provide adequate quantities of these nutrients.

The enrichment program has successfully accomplished its purpose. During the war years and for several years thereafter, symptoms due to a deficiency of the enrichment nutrients practically disappeared. In Newfoundland, an enrichment program contributed to the eradication of beriberi, to a 50 per cent decrease in infant mortality, decreases in the crude death rate and in tuberculosis. Nutritional deficiency states, once prevalent among the inhabitants of Chicago's Skid Row, have virtually disappeared. Kark et al., attributed this improved nutritional status to the enrichment program when a study of the food habits of the Skid Row population revealed one variable in their diet: enriched bread.

Avitaminoses, once prevalent among groups recognized as having poor diets, are now appearing in those who formerly had good eating habits. This finding reflects the shift, in our present-day economy, from food sources of the B vitamins, particularly thiamine. For our period of prosperity, Faith Clark has pointed out the need for emphasis on thiamine in nutrition education programs. The key to the role of enriched bread in such programs is revealed by a glance at its nutrient contributions: Six slices of enriched bread supply 23 per cent of the thiamine, 12 per cent of the calories, 18 per cent of the protein, 16 per cent of the calcium, 30 per cent of the iron, 16 per cent of the riboflavin, and 21 per cent of the niacin recommended by the Food and Nutrition Board as daily allowances for the twenty-five-year-old man.

References

1. Clark, Faith: Family Diets Today. Talk given at Nutrition Education Conference, Washington, D. C., April 1, 1957.
2. Kark, R. M., Vorhaus, L. J., Paynter, C. R., Morey, G. R., Imperiale, L., Sargent, F., Figueroa, W. G.: Lack of avitaminosis among alcoholics: Its relation to fortification of cereal products and the general nutrition status of the population. J. Clin. Nutrition, 1:3 (March) 1953.
3. McGrath, James M.: Revolution in Newfoundland —An Estimate of Public Health Benefits from Enriched Flour. Address at Tenth Anniversary Enrichment Luncheon, Hotel Pierre, New York, June 14, 1951.
4. Wilder, Russell M.: Baker's J., 16:6 (Feb.) 1953. 1953.

AMERICAN INSTITUTE OF BAKING

CAN YOU BE INSURANCE POOR?

Plans for Retirement

In planning for retirement or a "slowing down," the physician cannot include Social Security or a pension provided by an employer as his neighbor can. Therefore, the degree of his retirement depends upon his own planning during his active years.

The physician may feel it unnecessary to plan for retirement, calling to mind examples of elderly men, enjoying their continued activity and maintaining their reputations as competent practitioners years beyond the normal retirement age. It is an advantage of a profession, of course, that an individual can select his own retirement age—or can he? Unfortunately, compulsory retirement is not limited to employes of large firms, but in applying the term to the physician I am referring to another type of compulsory retirement, that brought on by failing health. Often such failing health occurs after disability income insurance has terminated. In discussing this problem recently, one physician said, "I don't know whether I shall want to retire, but if forced to retire, I want to be in a position to do so and still maintain my own independence and self respect." Doesn't that seem to be a sensible approach to the problem? That seems to be the feeling of many professional people.

It may seem discouraging to plan for retirement without the aid of Social Security or a pension with progressive income taxes making it more difficult to save and with increasing life expectancy following retirement. However, many physicians are not aware of some important tax factors written into the Internal Revenue Code of 1954. These tax factors eliminate entirely, or greatly reduce taxes on income at retirement. The three main factors are:

1. A new method for determining the taxable portion of annuity income.
2. A special retirement income credit for persons not covered by Social Security, and
3. Double personal exemptions for individuals age 65 or older. An example will illustrate how these tax credits offer real encouragement to planning a good retirement program.

In a previous article, an example was cited of a young doctor who set up a life insurance estate of $100,000 by purchasing $25,000 permanent life insurance and using a $75,000 term insurance rider. The $75,000 term rider is convertible regardless of health, in this case, within the ten years. This doctor probably will decide to make the $75,000 term or a good portion of it a permanent part of his estate by converting it when income permits. He may decide also to start planning a systematic retirement program. Using this policy as a stepping stone, he may build up as much assured life income as $12,000 per year at age sixty-five, without Federal Income Tax, if he and his wife are both sixty-five, because of tax credits afforded under the present tax code. If he is single, divorced, or a widower at sixty-five, he may have as much as $9,000 per year free of Federal Income Tax. The exact amount of life insurance or annuity income that one may have free of Federal Income Tax will depend on several factors.

As the physician approaches retirement and prepares to hand over his privileges and responsibilities in his chosen profession to his younger colleagues, he probably will list his financial objectives in this manner: (1) Adequate retirement income for as long as it will be needed, (2) adequate income for his widow if he should be the first to die and (3) the transfer of the unexpended estate to his children and grandchildren. In line with these objectives he probably will want his retirement income guaranteed for a minimum period of years for the benefit of his widow if he should die before the end of that period, but income continuing during his lifetime after the minimum period. An example of this is the income for 120 months certain but for life thereafter.

It may be advisable to have paid up life insurance adequate to cover estate clearance costs upon his death including administration costs and death taxes, and adequate to provide his widow with sufficient income should she survive him. With adequate lifetime income assured for himself and his wife, the physician may feel that he is in a position to transfer portions of his estate to his children while he is still living and be able to enjoy seeing the children and grandchildren benefit thereby. A further incentive to making such gifts may be found in the difference between the gift taxes and death taxes.

Certainly, it will be advisable for the physician to consult with his own life underwriter as well as his attorney and possibly the accountant and trust officer in mapping out the estate plan best suited for his particular situation. There is no one medicine or wonder drug that will cure all ills and there is no one estate plan that will solve the problem for everyone.

Whether it be the young physician beginning practice, the established practitioner or the doctor approaching retirement, who, would you say, is more likely to be "insurance or property poor"? The man who establishes financial goals and objectives early and is willing to earmark a reasonable portion of his earnings to achieve these goals by proper planning? Or the man who has not mapped out his financial plans in advance but is earmarking the same or a greater amount of his earnings for the general purpose of building an estate? The latter man likely will forget, too, that patients and financial programs both need regular check-ups.

The phrase "insurance poor" throughout has been used in its literal context. But, when someone says that Dr. A, in providing for his family's security, has become "insurance poor," would it not be appropriate more often to use the term "insurance rich?" He can say he is a wealthy man because he has, on one hand, peace of mind and, on the other hand, an unworried attitude toward the financial pressures of today. His plan has enhanced his family's standard of living instead of competing with it. If there were no such thing as insurance, he could not in conscience spend as freely as he does for fear of jeopardizing his own and his family's future.

HERBERT F. MISCHKE, C.L.U.

CONFESSIONS OF A HOBBYIST

Conclusion

The varied activities of which I have been writing have brought me infinite pleasure, and very little money. An occasional fee for a lecture on music, or an illustrated lecture on wild flowers

—an occasional sale of a pair of the little tables I've made in the shop, though most of these have been given as wedding presents to graduates of the Saint Paul Academy—a few dollars for written articles on radio—that is all. I instinctively shy away from definite orders to make things, partly because I don't like business transactions with friends, and partly because I don't want to sell anything that isn't perfect. But some of the activities have saved a lot of money around the house. For instance, the turning of an old barn into a summer place for my older son and his family made use of every bit of carpentry experience I had ever had, and some that I'd never had. There wasn't a right angle or a plumb line in the whole building, which made it a nightmare. Now it is a habitable house, with three bedrooms and a bathroom where the hayloft used to be, a modern kitchen in the old harness-room, and a pleasant living-room where the stalls used to be. Happily, I had another man helping me on this job—an old-timer, who knew more than I did about things like shingling, or fitting window-sash.

Certainly, what might be called "controlled versatility" enriches one's life. It also brings the painful realization that two things are rigidly rationed —time, and physical and mental energy. I quote from an address my father once made to the Yale Freshmen. " 'All things,' says the Emperor Marcus Aurelius, 'are implicated one with another, and the bond is holy: and there is hardly any thing unconnected with any other thing.' Thus there is scarcely a fragment that may not be fitted into the mosaic of life. . . ." Both the Emperor and Dean Briggs knew what they were talking about.

Life is full of curious surprises. The thing I believe I have done best and enjoyed most is the teaching of mathematics to adolescent boys, my unorthodox preparation having been a college course consisting mostly of classics and music! But into the mosaic of teaching, and of schoolmastering in general, almost every apparently unrelated hobby has come in somewhere. The reader may suspect, by this time, that I have no regrets for the hobbies. Now I must stop writing—there is a color print that doesn't satisfy me, and I must go to the darkroom and make it over!

JOHN DEQ. BRIGGS

AMERICAN HEART ASSOCIATION RESEARCH

The American Heart Association has announced it has channeled a total of $29,000,000 into research studies of cardiovascular diseases during its first ten years as a national voluntary health agency. More than $1.1 million of the total has been spent in Minnesota alone, according to the Minnesota Heart Association, one of fifty-six affiliates.

Marking its first decade of assault on diseases of the heart and circulation, American Heart Association is spending $7,000,000 on research during the current year alone. Twenty-five per cent of all funds raised by its affiliates are sent to national headquarters, which reallocates grants to scientists throughout the nation.

In Minnesota alone this year, fifty researchers at seven institutions are sharing $317,762 allocated from the 1957 Heart Fund drive. Sixty per cent of each Heart Fund dollar is spent on research, announced Dr. Milton Hurwitz, president of the Minnesota affiliate.

The American Heart Association plans a "massive assault" in the next decade on hardening of the arteries, which is responsible for more deaths and disability than any other ailment.

The 1958 Heart Fund drive is being conducted throughout the nation February 1-28. The goal in Minnesota is $505,000.

E. H.

ADDENDUM TO TUBERCULOSIS CASE FINDING PROGRAM FOR MINNESOTA

Your Tuberculosis Committee recently presented a seven-point program for tuberculosis case finding dealing primarily with tuberculin testing as the major substance of the program (see MINNESOTA MEDICINE, October 1957, page 732). It is feared by some that though the tuberculin test is definitely important, over-emphasis on this one procedure may encourage neglect of certain basic principles of case finding and control.

There is a saying, "Tuberculosis is where you find it." It would appear that many if not the majority of cases of active tuberculosis coming under care even today are first suspected by the private physician on the basis of some point other than the tuberculin test. Certainly any experienced physician will recall patients in his own practice discovered through application of tuberculin. He

will also recall cases in which suspicion was first aroused through a particular complaint heard in the office, home or hospital; or through some physical sign; or through an x-ray taken either for specific or general screening purposes; or through unexpected laboratory findings. Neither any one of these nor any other diagnostic hint of tuberculosis can safely be ignored by the conscientious physician or health advisor. On the other hand, such a point of suspicion does not by itself constitute a diagnosis nor even complete case finding. The isolated tuberculin reaction, questionable x-ray, or other finding suspended in mid-air and unsupported by other evidence is of itself rather fruitless. All too often in past surveys a person has been found with positive Mantoux reaction, x-ray finding or other significant sign of disease only to be hung up in a statistical exhibit with nothing further done, but any such point of suspicion which triggers a full case study may be health-saving or life-saving.

What constitutes such a plan of study? In brief outline, the following is suggested:*

1. A thoughtful history with searching questions regarding tuberculosis contact, symptoms, previous diagnoses, former positive or negative tuberculin reactions, x-ray study, operations, etc.

2. Comparable physical search for tuberculous foci.

3. Meticulous application and interpretation of the Mantoux test, and perhaps later re-application.

4. Judicious x-ray filming, routine or special, of the chest or other body areas under suspicion.

5. Competent bacteriological study of body secretions, fluids, and tissue specimens by microscopic, and more particularly by cultural and animal inoculation methods.

Any one of the above procedures may constitute a trigger mechanism for case study but only the combination of procedures constitutes balanced case finding.

In all such study we must remember that no one test is completely infallible in demonstrating tuberculous disease. The finding of tubercle bacilli has in the past been considered positive proof, but today bacteriologists occasionally doubt their own findings. Over-reliance on the x-ray film misleads us fairly frequently and the tuberculin reaction can likewise be contradictory though far less often. Yet one sanatorium in the State has had several proven cases of active tuberculosis in recent years in which tuberculin reactions were negative during at least part of the period of activity.

This is not to minimize the real value of such test procedures and survey methods as we have. As we narrow down the incidence of tuberculosis today, these procedures need to be applied more widely and strategically than before. The ideal case finding survey still would consist of the following steps in order:

1. Mantoux testing of all those not previously known to be positive reactors.

2. Thorough periodic clinical review and chest x-ray filming of all positive reactors and known cases throughout their lifetime.

3. Bacteriologic, special x-ray and other special laboratory examinations as clinical study may indicate.

4. Faithful reporting to the State Department of Health of all known cases of tuberculosis.

Unfortunately, we cannot routinely or even frequently reach our public health aim directly; we are forced into detours. In the present instance a practical answer is to reach annually as many in the community as possible but in particular to aim at (1) groups with known high incidence of tuberculosis by virtue of racial, economic, social, occupational or other factors, or known contact to active cases (this should include special investigation of the record of any case first reported by death certificate; also thorough study of the contacts of such cases); (2) groups known to be of special epidemiologic concern to the rest of society, such as food handlers and those occupationally close to children and adolescents.

Our plea then should be for a balanced program, a broad and persistent one, attacking at all vulnerable points, not only at one or two. And finally, to remember that there is no substitute in tuberculosis case finding for the alert physician; for the man with a high index of suspicion, the man so thorough as to be dissatisfied with the performance of one isolated test unsupported by broader case study.

R. W. Backus, M.D.

*For fuller outline, see Report of Council on Public Health reprinted in Minnesota Medicine, Feb., 1957, Page 137.

President's Letter

MEDICAL PRACTICE IN A CHANGING WORLD

Immersed in a busy practice, we doctors presume that the circumstances under which we practice are unchanging and, in fact, unchangeable. The private practice of medicine, whether alone or in a group, in this country has seemed the ideal way to care for the sick and to improve the health of those who are apparently well. In the United States, the freedom of the sick to choose his own physician has been a jealously defended right. Conversely, the freedom of the physician to choose his patients and his type of practice without dictation or interference by third parties has been a foundation on which American Medicine has been built.

These freedoms have been lost in several foreign countries and are being attacked in this country. Their value is unquestioned by those who remember the principles on which our government and our culture were founded, but there are many who now would change our way of living. The medical profession has been called conservative, not to mention reactionary, in its efforts to maintain freedom of choice, but the most liberal political idea ever proposed is the American idea of personal, individual freedom of choice in personal, individual matters. The care of one's personal health and the climate of medical practice are basic factors in the general freedom. Change in these basic factors is a major step toward change in our whole way of living.

As we carry on our practices and try to keep up with the demands of our patients and the advances in medicine, we feel that nothing more can be expected of us. Knowing our way of practice is the best, we expect it not to change; however, it can change and is changing. How radical the change may be is not determined only by ourselves or by our patients, but by the general pattern of political and economic life. It is determined by the County Commissioner and the County Welfare Boards and management-labor contracts. It is determined by the State Government, the State Welfare Boards, the State Legislatures, through the State Income Tax Commissioner, the State Board of Medical Examiners, and the medical practice acts. It is determined by the insurance companies, the Blue Shield, and perhaps by co-operative clinics and other "third parties" to medical practice. It is also determined by the Federal Government through Medicare, the Veterans Administration, Social Security, the Armed Services and even the Department of Agriculture through the Food and Drug Administration.

Our relations with all these forces are not individual. They can only be carried on through the profession as a whole; more specifically, only through our County, State and American Medical Associations. Our Medical Associations can do for us what we want only if we, individually, tell them what we want. That can be done only when we are there to discuss, to vote and to elect. The voice of the profession may be the president of the society, but the policy is made by the boards of directors of the County Societies and by the delegates to the State Association and by their delegates to the American Medical Association.

Election time is here. Choosing a director or a delegate is serious business. When you find a good one, keep him if you can, because he is the one who decides policy, who is the power in the association and is part of the group which deals with those who determine how you shall practice; talk to him, know him, know what he and the societies are doing, and then support him.

Horatio R. Sweetser

President, Minnesota State Medical Association

Medical Economics

Edited by the
Committee on Medical Economics,
Minnesota State Medical Association
George Earl, M.D., Chairman

MEDICARE PROGRAM IS ONE YEAR OLD

The military's medicare program was one year old on December 7. To date, according to the Defense Department, the government has paid more than 300,000 physicians' bills amounting to $22 million, and over 200,000 civilian hospital bills totaling $21 million. While the combined total is considerably under the estimated $76 million a year for the program, the Office of Dependent's Medical Care points out that there is a backlog of claims. In all likelihood, the President's budget going to Congress next month will ask for around $76 million.

Administrative costs, according to OCDM, have been running around 3% of total expenditures. Almost 40% of medicare patients have been maternity cases. The Air Force leads the services with 41% of eligible dependents participating; then the Navy, with 32%; Army, 25%, and Public Health Service, 2%.

The medicare program was enacted into law on June 7, 1956, and became effective six months later.

OCTOBER BUSY MONTH FOR FDA IN ENFORCING LAWS

The Food and Drug Administration reports that in October of 1957: 121 voluntary actions to comply with the law; seizure of 561 tons of contaminated foods in 96 Federal court actions; convictions in nine cases involving illegal over-counter sale of prescription drugs; one court conviction and granting of injunctions in two cases involving interstate shipment of insanitary foodstuffs and certain self-treatment devices. Included in latter category was a vibrator which was represented as a treatment for cancer and heart ills.

UNCLE SAM, M.D., PROSPERS AS HE PRACTICES

Nearly one-half of all medical costs are paid for with tax money, states Dr. Thomas H. Alphin, of the AMA's Washington office.

"The physicians in this country can no longer afford to scorn their civic duty and duck their responsibilities to be part of the Federal government," he declares.

Here's why. The amount proposed to be spent for medical care and research in 1957-58 is $3 billion, more than 10 per cent of the non-defense Federal budget and over 25 per cent of the total medical budget of the whole country, including all costs, physicians' fees, hospital bills, drugs, hot water bottles—the whole ball of wax.

And, says Dr. Alphin, when the state and local government spending is added to this amount it is almost double—nearly one-half of all medical costs are paid for with tax money: $6 billion a year total, half Federal, half state and local, and amounting to half the Nation's entire medical cost!

Biggest spenders for medical care: the Vets Administration, the Dept. of Defense, and the Dept. of HEW. "They digest dollars at the rate of $6½ million a day, $250 million per hour, nearly $75.00 every time a clock ticks!"

With the beginning of increased vendor payments to OASI benefits, Dr. Alphin states, the medical care expense of the Federal government will be increased by possibly as much as $150 million annually, this money to be matched by the states, for a resounding total of $300 million a year.

Tax dollars are not the only concern of harassed physicians. There are millions of people—patient—involved in the story of Uncle Sam, M.D.

Says Alphin: "Legislation currently proposed would provide for partial Federal payments for sickness and injury of Federal employes and their dependents, about seven million in addition, which would make a total of nearly 50,000,000 people entitled to free government care to some degree."

SOCIAL SECURITY—A GIFT HORSE?

Social Security is in trouble. Benefit funds are rapidly depleting as payments exceed federal estimates. By 1959 expenditures in excess of income may be anticipated. Deficits will be covered by higher taxes.

Rates of employer-employe Social Security participation are expected to more than double by 1975, from the two per cent contributions in 1956 to four and one-quarter per cent by 1975.

Rates for self-employed participants will increase to six and three- eighths by 1975. Currently the rate is three and three-eighths per cent.

The following table indicates the anticipated Social Security rate increase based on $4,200 annual wage.

Calendar Year	*Employe*	*Employer*	*Self-Employed*
1956	2 %	2 %	3 %
1957-59	2¼%	2¼%	3⅜%
1960-64	2¾%	2¾%	4⅛%
1965-69	3¼%	3¼%	4⅞%
1970-74	3¾%	3¾%	5⅝%
1975 and after	4¼%	4¼%	6⅜%

SHARP RISE NOTED IN HOSPITAL COSTS

The *Monthly Labor Review,* in a report on medical care costs, in the cost of living index, notes that in the past twenty years hospital costs have risen sharply in contrast to physicians' fees. The Bureau of Labor Statistics lists these increases between 1936 and 1956:

Hospital room rates	264.8%
Dentists' fees	82.1%
General Practitioners' fees	72.8%
Surgeons' fees	59.5%

In the same period, medical care costs generally have lagged behind costs of food, personal care other than medical and clothing. The report makes this observation: "With the higher level of living attained in 1950, relative expenditures for medical care tended to increase as incomes increased, as is usually true of items considered as 'necessities' in the family budget. The fact that this pattern has begun to appear in the spending of workers' families indicates the high order of importance they place on medical care. . . ."

THE NEW VA ADMINISTRATOR

Sumner G. Whittier, a former lieutenant governor of Massachusetts and an official of the Veterans Administration since last January, has been appointed chief of the agency, succeeding Harvey V. Higley who resigned recently. Mr. Whittier has been VA's chief insurance director since last January; that department handles policies covering over 6 million veterans. A Republican, Mr. Whittier was elected to his first post in 1938 as member of the Everett (Mass.) Common Council, three years after graduating from Boston University. He served in the state legislature, was in the Navy during World War II and was lieutenant governor from 1953-1956.

MEDICARE PATIENTS NOW REQUIRE IDENTIFICATION CARDS

All dependents of service personnel, with minor exceptions, are now required to present uniformed services identification and privilege card when they apply for medical service. The form number is DD Form 1173 and a photograph of the dependent appears on the reverse side. Exceptions to strict enforcement are as follows:

1. In cases of acute medical, surgical or emotional conditions of an emergency nature requiring immediate medical care for humanitarian reasons.

2. In cases of children under ten years of age. Cards have not been issued to these children and eligibility for care must be established by parents or guardian to the satisfaction of the physician.

3. In cases where, for special reasons connected with turnover of service personnel, the dependent does not possess a card. In such a case, according to the government bulletin, it will be necessary to rely on some other means of identification. It is obviously anticipated that most eligible dependents over ten years of age will be supplied with the required card.

In all cases other than those involving children under ten, the dependent or service sponsor should be required by the physician to show on the physician's claim form, DA Form 1863, in items 6 and 7 (or on an attached statement) the type of documentation or identification form used.

Only wives, dependent husbands and legitimate children of uniformed service members are eligible for care under the program.

HOW AMA DOLLARS ARE SPENT

At a recent meeting in Miami, Dr. George F. Lull, AMA secretary-general manager, told an audience how the AMA spends its annual income of approximately $10 million. The AMA's main sources of revenue are from dues and subscriptions which amount to $5,192,000, and from advertising which brings in excess of $4 million. Some expenses reported to the nearest thousand are: public relations, $400,000; Council on Medical Education and Hospitals, $376,000; Bureau of Health Education, $296,000; Washington Office, $227,000; Council on Medical Service, $206,000; Membership Records, $202,000; Bureau of Medical Economic Research, $173,000; Biographical Records, $155,000; American Medical Education Foundation Overhead, $119,000; Law Depart-

ment, $111,000; Bureau of Exhibits, $109,000; Council on Drugs, $99,000; Council on Industrial Health, $76,000; Council on Rural Health, $68,000; Council on Medical Physics, $61,000, Council on Foods and Nutrition, $56,000.

THE MEDICAL PROFESSION PLAYS IMPORTANT ROLE IN 85TH CONGRESS

Numerous issues of interest and importance to the medical profession are set for action during the second session of the 85th Congress.

The Jenkins-Keogh pension plan bill (HR 9-10) is scheduled for late January hearings. Senator William Proxmire (D., Wis.) will introduce a bill embracing many features of Forand bill (HR 9467), including free hospitalization for social security recipients. Medical training is expected to receive considerable attention.

Congress is also expected to examine Medicare and decide whether any legislative changes in this program of medical services for military dependents are indicated. Also docketed for later consideration is the question of providing medical and hospitalization insurance for Federal employes and their dependents.

Expenditures for Hill-Burton hospital expansion, financial aid for medical research and contributions in support of vocational and medical rehabilitation of the physically handicapped will be included in public hearings by House Government Operations Subcommittee on Intergovernmental Relations into Federal grants for states and municipalities.

Also expected in the session, is the report and recommendations of the House Government Operations Subcommittee on Legal and Monetary Affairs regarding advertising controls for weight-reducing drugs and filter cigarettes.

SEARS-ROEBUCK FOUNDATION OFFERS AID IN DOCTOR PLACEMENTS

The Sears-Roebuck Foundation has announced a program aimed at helping communities who have no doctor to obtain the services of a doctor. The new program will stress the building or remodeling of a medical center that is either better or equal to those available in the city. Ever since the physician loan program had to be dropped, the Foundation has been working on another project in the field of medical distribution.

The Foundation has arranged with an architect to provide plans for an economical and functional medical center which is adaptable to local building materials. This building incorporates every modern feature necessary for quality medicine. It is furthermore designed to simplify the actual work load of the help by an efficient office arrangement.

The Medical center is designed to provide outpatient service where needed and can be easily expanded from a one to a two doctor unit. The cost of this building fully equipped except for examining tables, x-ray, and waiting room chairs ranges from $12,000 to $25,000. The Foundation will also be prepared to provide architectural advice on remodeling if the building is attractive and suitable.

The Foundation is prepared to provide any community so chosen to participate in this program the following: 1. Assist in conducting an economic survey of community to see if area can financially support a doctor. 2. Consultation and advice on fund raising and organizing the community. 3. Complete blueprints and building specifications on the medical center or advise on remodeling depending on which is most feasible. 4. Our services along with the AMA and our Medical Advisory Board to aid in obtaining a doctor.

Communities chosen to participate must be approved by the state society and be able to support a physician. A major consideration is whether a community is willing to help itself and attempt to raise it's own funds. Any community willing to do so regardless of its proximity to areas or medical need would qualify under this program.

If you should desire any additional information, write to Mr. Norman H. Davis, Director, The Sears-Roebuck Foundation, 3333 Arthington St., Chicago, Illinois.

U. S. EXPECTS PHYSICIAN-DENTIST SUITS

Recently the U. S. Court of Claims ruled in favor of an ex-Army dentist who, having served involuntarily as a private in 1953-54, was suing for back pay of a Dental Corps captain. Unanimous decision was that the Army flouted the doctor-draft law by using the man in his professional role while keeping him on private's pay. Court held that the military was within its rights in denying him a commission because of Communistic affiliations but erred in giving him officer's responsibility.

(Continued on Page A-57)

Medical-Legal Opinions

By JULE M. HANNAFORD, Legal Counsel
Minnesota State Medical Association

CHANGE MADE IN INDIVIDUAL INCOME TAX FORMS

Late in October, 1957, the Internal Revenue Service announced that a new line had been included in Form 1040, which is the form prepared for use by individual taxpayers in reporting their income taxes. Line 6(a) requires the listing of "Travel, reimbursed expenses, etc.," which amount is subtracted from income reported in line 5. The Instructions, which are prepared by the Internal Revenue Service for use in connection with Form 1040, explain that an employe should include in line 5 all payments he receives as reimbursement for expenses he has incurred and should deduct the expenses listed in line 6(a).

Most physicians in preparing and filing their income tax returns are in no way affected by this change. The change applies only to expenses incurred and reimbursements received by *employes*. Physicians in private practice should report the earnings and expenses of their practice in Schedule C (Form 1040) while the income and expenses of a medical partnership must be reported in Form 1065. However, some physicians receive income from their medical services in part or in whole as employes, such as physicians employed by a medical association or clinic. A physician whose income taxes are withheld from his salary and who receives a W-2 statement from his employer at the end of a year receives income as an *employe*. The change in Form 1040 would affect the preparation of tax returns by such physicians.

While physicians who are in private practice and are not employes of a medical clinic or association need not be concerned with the change made in Form 1040, a similar problem exists in determining what types of payments must be included by an individual practitioner in line 1 of Schedule C (Form 1040) as "Total receipt," or by a medical partnership in line 1 of Form 1065 as "Gross receipts." The Instructions printed on the back of Schedule C (Form 1040) state:

> "Line 1—Total Receipts.—Include all income derived from your trade or business."

Physicians on occasion bill patients for certain expenses in addition to their normal fees. For instance, a physician may find it desirable to bill a patient separately for travel, lodging, and meal expenses which are incurred in connection with services performed outside of the physician's community. Such expenses may be separately billed or shown as a separate item on a fee statement. If a physician charges a *lump sum* to cover such expenses, which is not based on the exact expenses incurred, he must include the payment he receives in his "Total receipts" ("Gross receipts" in the case of medical partnerships), and deduct the expenses which are actually incurred. On the other hand, if a physician bills a patient for the exact amount of the expenses which he has incurred and subsequently receives *reimbursement* therefor, he need not include such reimbursements in his "Total receipts." This follows both because such payments have not been made to a physician by his employer and because payments received as reimbursements of such expenses may be considered as repayments of a loan rather than as income. The same result applies to *advancements* received by a physician from a patient to cover business expenses to be incurred by the physician, where any amounts not spent for such expenses are returned or credited against the patient's bill. Of course, if in either of such instances a physician does not include the payments in his gross income, he cannot deduct the expenses. No guaranty can be made that in the future the Internal Revenue Service will not require advancements and specific reimbursements to be included in "Total receipts."

To avoid the necessity of reporting as income in tax returns payments of expenses received from patients and then detailing in tax returns the actual expenses, physicians may find it desirable to bill a patient for the exact amount of certain expenses, rather than to bill the patient an amount as an expense allowance in estimation of the expenses or to increase the bill of the patient to cover the expenses. However, the fact that such payments do not have to be reported does not mean that complete records showing the incurrence and allowability of the expenses should not be kept. If a physician's tax return should be audited, he might be requested by the Examining Revenue Agent to produce his bank statements. The Agent may ask why certain deposits were not included in the physician's income tax returns. At that time it will not be enough for the physician to state that the payments were received as reimbursements or advances for expenses; the physician will have to be able to back up his statements with proof. For that reason all physicians should strive to keep complete and detailed records. These records should itemize separately the expenses for which the physician is reimbursed, such as hotel bills, meals, train or plane fare, etc., and the reason the expenses were incurred. In addition, documentary evidence of the larger expenditures should be saved, such as ticket stubs, receipts for hotel bills, etc. Only by following such a procedure is a physician completely protected if his returns are audited.

Committee Action

The Child Health Committee

National PTA Medical Care
American Public Health Association

In May of 1956 the National Congress of Parents and Teachers adopted a series of recommendations entitled "The Periodic Health Appraisal for Well Children from Birth Through High School." Dr. Henry Helmholz, formerly of the Mayo Clinic, and a past president of the American Academy of Pediatrics, is health chairman for the National P.T.A. He was assisted in the development of these recommendations by representatives of about twenty different organizations including the American Academy of General Practice, the American Academy of Pediatrics, the American Academy of Ophthalmology and Otolaryngology, the American Medical Association, the American Dental Association and the American Psychiatric Association.

This program was developed as a logical extension of the so-called "Summer Round-Up" which the P.T.A. has sponsored since 1925. The Summer Round-Up is the familiar program of health examination for children about to enter school for the first time. The new program calls for continued health supervision for all children. The groups involved in formulating this program repeatedly emphasize that the continued health supervision should be done where possible by the physician and dentist who normally serve the child and family.

National P.T.A. Recommendations

1. That the National Congress of Parents and Teachers adopt a policy supporting and encouraging a program of continuous health supervision of children from birth through their school experience, rather than only a program of single appraisal on school entrance.

2. That the National Congress of Parents and Teachers recommend to its local units a promotional and educational program that will tend to bring children and their parents into effective contact with the health resources of the community.

3. That wherever possible, these should be the physician and dentist who normally serve that child or family, whether they be working in private practice or in a community health service.

A program of continuous health supervision has been recommended repeatedly by medical, dental, school, and public health personnel. The role of the parent-teacher asociation will be to acquaint parents with the means of utilizing effectively the resources available to meet the health needs of children, beginning at birth and continuing through the school years. The parent-teacher association also should work with the professions and agencies of the community in planning and carrying out such a program.

A.P.H.A. Recommendations

A program of the character contemplated has been outlined by a committee of the American Public Health Association, in a report entitled Health Supervision of Young Children, the essential elements of which are as follows:

A. Periodic health appraisal of the child, preferably by his own physician and dentist, to give long-term continuity, including—
   1. Well-recorded medical, dental, and developmental history
   2. Physical examination to evaluate physical, mental, and emotional development and adjustment, and to discover deviations

B. Immunizations

C. Consultation with parents
   1. To advise about feeding, nutritional needs, immunizations, recommended corrective procedures, accident prevention, and health protection.
   2. To interpret what a child is like—his normal growth and his particular state of development
   3. To assist in the management and prevention of behavior and personality problems
   4. To plan required treatment and to refer parents to appropriate services when necessary
   5. To help parents become more capable and self-confident, and to build good parent-child relations and promote family well-being

D. That the National Congress of Parents and Teachers recommend to its local units that the following activities, among others, be accepted as their role in the child health program
   1. *Educational*
      (a) Acquaint residents of local communities with existing health facilities and explain how to use them effectively
      (b) Interest school personnel in the importance of preschool health as preparation for school attendance
      (c) Aid in bringing about realization of the importance of the public health unit, especially the nurse, in health promotion

(Continued on Page A-42)

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul 2, Minnesota

F. H. Magney, M.D.
Secretary

PHYSICIANS LICENSED FEBRUARY 13, 1957

January, 1957, Examination

Name	*School*		*Address*
BERZINA, Ligita Gina	U. of Minnesota	MD 1955	1800 S. Virginia St., Hopkinsville, Ky.
BROWN, Russell Thomas	Marquette U.	MD 1956	Browerville, Minn.
CHARTIER, Yvon	U. of Montreal	MD 1954	200 1st St. S.W., Rochester, Minn.
CLIMIE, Andrew Robert Wilson	U. of Glasgow, Scotland	MB 1950	200 1st St. S.W., Rochester, Minn.
GRIFFIN, Arthur Russell	U. of Wash., Seattle	MD 1955	Mpls. Gen. Hospital, Minneapolis, Minn.
GUENTHER, Dean Edward	U. of Wash., Seattle	MD 1956	42 E. Orme, West St. Paul, Minn.
HAYWARD. Mark Alan	U. of Manitoba	MD 1952	Gackle, N. D.
HENRIE, Joyce Elaine Rich	U. of Utah	MD 1955	200 1st St. S.W., Rochester, Minn.
JOHNSRUDE, Irwin Stanley	U. of Manitoba	MD 1956	Fairmount, No. Dak.
KING, Harold E.	U. of Wash., Seattle	MD 1955	200 1st St. S.W., Rochester, Minn.
KOLARS, Charles Paul	St. Louis Univ.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
LAPP, Maurice M.	Northwestern U.	MD 1956	Miller Hospital, St. Paul, Minn.
LOGOTHETIS, John Achilleas	U. of Thessaloniki, Greece	MD 1950	U. of Minn. Hospitals, Minneapolis, Minn.
LU, Cheng-en	West China Union U., Cheng-tu, China	MD 1949	3312 First Ave. S., Minneapolis 8, Minn.
MIGAKI, Hoyoko	U. of Oregon	MD 1954	Mpls. Gen. Hospital, Minneapolis, Minn.
MOLLER, Jurgen	Christian-Albrechts U., Kiel, Germany	MD 1952	331 Oak St., Farmington, Minn.
MORAN, Walter Harrison	Harvard U.	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
MULHAUSEN, Robert Oscar	U. of Illinois	MD 1955	Vet. Adm. Hospital, Minneapolis, Minn.
MUNRO, Jerrold Albert	U. of Minnesota	MD 1955	4719 Townes Road, Edina, Minn.
NORSTROM, Craig Wilbur	U. of Alberta	MD 1955	200 1st St. S.W., Rochester, Minn.
PASH, Earl Leon	U. of Manitoba	MD 1950	U. of Minn. Hospitals, Minneapolis, Minn.
READ, Raymond Charles	U. of Minnesota	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
SCOTT, Alan Brown	U. of California	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
SMITH, Jr., Lester Leigh	Med. Col. of S. Car.	MD 1953	200 1st St. S.W., Rochester, Minn.
THAL, Alan Philip	U. of Cape Town, S. Africa	MB, Ch.B. 1949	U. of Minn. Hospitals, Minneapolis, Minn.
TROTMAN, Neil McKay	U. of Illinois	MD 1956	1698 Ford Pkwy., St. Paul 16, Minn.
UTZ, Philip Hoy	U. of Michigan	MD 1953	La Crescent, Minn.
WEINSTEIN, Marvin Jerome	U. of Oregon	MD 1955	200 1st St. S.W., Rochester, Minn.
WISE, James Kearney	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
BACHHUBER, Max Otto	U. of Wisconsin	MD 1932	Alma, Wis.
BARRIER, Jr., Charles Wesley	U. of Texas	MD 1954	200 1st St. S.W., Rochester, Minn.
BRUNGARDT, Bernard Aloysius	Creighton U.	MD 1946	200 1st St. S.W., Rochester, Minn.
ENGSTROM, Jr., Perry H.	U. of Pittsburgh	MD 1947	403 7th St. N., Wahpeton, No. Dak.
GILES, Jr., William Fisher	Creighton U.	MD 1955	200 1st St. S.W., Rochester, Minn.
HARRIS, James Roe	U. of Wisconsin	MD 1951	Gen. Delivery, Winston-Salem, N. C.
HOLBERT, James Allen	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.
JACKSON, J. Albert	U. of Pittsburgh	MD 1948	U. of Minn. Hospitals, Minneapolis, Minn.
KEEFE, William Peter	U. of Maryland	MD 1955	200 1st St. S.W., Rochester, Minn.
LARSON, Norman Eugene	Northwestern U.	MD 1953	200 1st St. S.W., Rochester, Minn.
LINKE, Charles Allen	U. of Illinois	MD 1950	205 W. 2nd St., Duluth, Minn.
McALLISTER, James	U. of Iowa	MD 1953	200 1st St. S.W., Rochester, Minn.
McGOON, Dwight Charles	Johns Hopkins U.	MD 1948	200 1st St. S.W., Rochester, Minn.
MITCHELL, William Charles	U. of Louisville	MD 1952	200 1st St. S.W., Rochester, Minn.
NEIL, Robert Lewis	U. of Tennessee	MD 1951	200 1st St. S.W., Rochester, Minn.
NEUMANN, Jr., Roland Ferdinand	Washington U., St. Louis, Mo.	MD 1944	1935 Med. Arts Bldg., Minneapolis 2, Minn.
NIEHAUS, Karl Friedrich	U. of Nebraska	MD 1953	200 1st St. S.W., Rochester, Minn.
PETERSEN, Byron Douglas	U. of Nebraska	MD 1947	U. of Minn. Hospitals, Minneapolis, Minn.
PORTELA-MORALES, Angel Ismael	Med. Col. of Va.	MD 1951	200 1st St. S.W., Rochester, Minn.
REICHELDERFER, Thomas Elmer	Johns Hopkins U.	MD 1950	Rm. 11N322, Bldg. 10, Nat. Inst. of Health, Bethesda 14, Md.
RICE, William Henry	U. of Nebraska	MD 1956	825 S. Oak St., Owatonna, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Name	*School*		*Address*
ROBERTS, George Albert	U. of Illinois	MD 1944	St. Olaf's Hospital, Austin, Minn.
SMITH, John Hough	U. of Michigan	MD 1955	200 1st St. S.W., Rochester, Minn.
WARVEL, Jr., John Henry	Johns Hopkins U.	MD 1955	200 1st St. S.W., Rochester, Minn.
WEBB, Jr., Robert Basye	U. of Virginia	MD 1952	200 1st St. S.W., Rochester, Minn.
WEISSMAN, Fredrick	Wayne Univ.	MD 1953	200 1st St. S.W., Rochester, Minn.
WILLIAMS, Hugh Jones	U. of West Ontario	MD 1950	200 1st St. S.W., Rochester, Minn.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
ADDISON, Robert Meade	Northwestern U.	MD 1953	200 1st St. S.W., Rochester, Minn.
BOYD, Frank Elmer	Harvard U.	MD 1953	Jasper, Minn.
ENGEL, Andrew George	McGill Univ.	MD 1955	Nat. Cancer Inst., Nat. Inst. of Health, Bethesda, Md.
FRANCKOWIAK, John Joseph	Georgetown U.	MD 1955	200 1st St. S.W., Rochester, Minn.
HAMILTON, Michael Joseph	Creighton U.	MD 1952	200 1st St. S.W., Rochester, Minn.
JARCHOW, Brian H.	Loyola U.	MD 1953	200 1st St. S.W., Rochester, Minn.
LARSON, Robert LeRoy	Geo. Washington U., Washington, D. C.	MD 1953	200 1st St. S.W., Rochester, Minn.
OKIHIRO, Michael Masaru	U. of Michigan	MD 1955	200 1st St. S.W., Rochester, Minn.
ROSS, Helen Margaret	Northwestern U.	MD 1955	200 1st St. S.W., Rochester, Minn.
STERLING, Harold Melvin	Yale Univ.	MD 1951	U. of Minn. Hospitals, Minneapolis, Minn.
TASWELL, Howard Filmore	New York Univ.	MD 1953	200 1st St. S.W., Rochester, Minn.
WILBUR, Jr., Oscar Milton	Boston Univ.	MD 1946	Hibbing Gen. Hospital, Hibbing, Minn.

PHYSICIANS LICENSED MAY 24, 1957
April, 1957, Examination

Name	*School*		*Address*
BERGER, Saul	U. of Toronto	MD 1943	901 Boyd Bldg., Winnipeg, Man., Can.
BLEDSOE, Francis Henry	Med. Col. of S.C.	MD 1953	200 1st St. S.W., Rochester, Minn.
CAMPBELL, Mervin Willard	Northwestern U.	MD 1956	8821 Jenrich Ave., Midway City, Cal.
DAVIDSON, William David	Duke Univ.	MD 1953	St. Joseph, Minn.
ENGLAND, Rodney Wayne	U. of Illinois	MD 1956	1714 E. Brown St., Springfield, Ill.
FRANK, Gilbert Sherman	U. of Minnesota	MD 1954	1807 Kentucky Ave., St. Louis Park, Minn.
HATHAWAY, Walter Ryland	Marquette U.	MD 1956	531 N. Main St., South Bend, Ind.
HEINZ, John N.	Loyola U.	MD 1956	109 Lincoln Ave. S.W., Wadena, Minn.
HO, Shu Kang	Army Med. College, Chekiang, China	MB 1947	1700 University Ave., St. Paul 4, Minn.
JACKSON, Carl Robert	Jefferson Med. Col.	MD 1956	1027 E. Johnson St., Madison, Wis.
JUDGE, Crawford MacCullough	U. of Manitoba	MD 1951	5584 Harvard Place, Ontario, Cal.
LEES, Jack Ramon	Northwestern U.	MD 1956	1328 2nd So., Fargo, N. D.
MUHICH, Donald Francis	Harvard U.	MD 1956	513 Grant Ave., Eveleth, Minn.
PETERSON, John Robert	U. of Oregon	MD 1956	Wisdom, Montana
SNYDER, Joseph	U. of Wash., Seattle	MD 1955	301 W. 37th St., Topeka, Kansas
TAYLOR, Gordon Chris	Washington U. St. Louis, Mo.	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
WHITE, James Edwin	U. of Oklahoma	MD 1954	Vet. Adm. Hospital, Minneapolis, Minn.
YOUNG, Hadley Rasch	Duke Univ.	MD 1956	929 Med. Arts Bldg., Duluth 2, Minn.
ZUPAN, George	U. of Oregon	MD 1956	4036 S. 6th St., Klamath Falls, Ore.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
AIGNER, Bruno Robert	St. Louis U.	MD 1952	200 1st St. S.W., Rochester, Minn.
EVENSON, David Johann	Stanford U.	MD 1954	200 1st St. S.W., Rochester, Minn.
GATZKE, Laurence Duane	U. of Iowa	MD 1955	200 1st St. S.W., Rochester, Minn.
GRISMER, Jerome Theodore	U. of Cincinnati	MD 1948	4959 Excelsior Blvd., Minneapolis 16, Minn.
HANSKE, Edward Albert	U. of Iowa	MD 1947	402 Phys. & Surg. Bldg., Minneapolis Minn.
KOLP, Berton Andrew	U. of Texas	MD 1950	105 E. Minn. Ave., Glenwood, Minn.
LEPPINK, Harold Bernard	Wayne Univ.	MD 1949	Two Harbors, Minn.
LIEBER, Arthur	U. of Louisville	MD 1953	Vet. Adm. Hospital, Minneapolis Minn.
OTTO, Evan Louis	Northwestern U.	MD 1951	Vet. Adm. Hospital, Vancouver, Wash.
PERSON, Douglas Allen	U. of Michigan	MD 1955	204 Med. Arts Bldg., Albert Lea, Minn.
REED, Henry Hofmann	U. of Kansas	MD 1953	205 W. 2nd St., Duluth, Minn.
REITER, Benjamin Reynolds	Harvard U.	MD 1934	U. of Minn., Stud. H.S., Minneapolis, Minn.
ROSS, Jr., James Vincent	Duke Univ.	MD 1955	200 1st St. S.W., Rochester, Minn.
SUMMAR, Marion Thomas	Tulane Univ.	MD 1949	727 Grant Ave., Virginia, Minn.
SWANSON, Wallace Leroy	Bowman Gray Univ.	MD 1956	3017 Bloomington Av. S., Minneapolis, Minn.
TAMA, Lawrence	U. of Penna.	MD 1953	200 1st St. S.W., Rochester, Minn.
WILBUR, III, Dwight Locke	Stanford U.	MD 1955	200 1st St. S.W., Rochester, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
BOOLUKOS, George Peter Nicholas	Albany Med. Col.	MD 1953	200 1st St. S.W., Rochester, Minn.
BRADLEY, John Grover	Geo. Wash. U.	MD 1951	629 Med. Arts Bldg., Minneapolis 2, Minn.
FRIEDLIEB, Oskar Peter	American U. of Beirut	MD 1948	2215 Plymouth Ave. N., Minneapolis, Minn.
MOLLER, Julia Clara	Med. Col. of Va.	MD 1953	1823 E. 5th St., Duluth, Minn.
PETERSON, Clifton Edward	U. of Rochester	MD 1954	200 1st St. S.W., Rochester, Minn.
STEVENS, William Augustus	New York Med. Col.	MD 1955	Vet. Adm. Hospital, Minneapolis, Minn.

PHYSICIANS LICENSED JULY 12, 1957

June, 1957, Examination

Name	*School*		*Address*
ANDERSON, Allen Merle	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
ANDERSON, Daniel Clarence	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
BAAK, William W.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
BACH, Jerome Menuhim	U. of Minnesota	MD 1957	USPHS Hospital, Baltimore, Md.
BALIAN, Lucy June	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
BARNARD, Jr., George Linal	U. of Minnesota	MD 1953	U.C.L.A. Med. Center, Los Angeles, Cal.
BAUDER, George B.	U. of Minnesota	MD 1957	Asbury Meth. Hospital, Minneapolis, Minn.
BEALKA, Richard James	St. Louis U.	MD 1956	St. Joseph's Hospital, St. Paul, Minn.
BEGGS, John Harkness	U. of Minnesota	MD 1957	Gorgas Hospital, Ancon, Canal Zone
BELAU, Paul Garnett	U. of Minnesota	MD 1957	USPHS Hospital, Staten Island, N. Y.
BEN-ORA, Avi	U. of Minnesota	MD 1957	San Joaquin Gen. Hosp., French Camp, Cal.
BERENDES, Heinz	U. of Munich, Germany	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
BERGSTROM, LaVonne B.	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis, Minn.
BERMAN, John Alexander	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
BINGHAM, George Conley	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
BRADLEY, William Edward	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
BRENK, Paul E.	U. of Minnesota	MD 1957	U. S. Army Hospital, Fort Benning, Ga.
BROOKS, Bancroft Morgan	U. of Minnesota	MD 1957	Kaiser Fdtn. Hospital, San Francisco, Cal.
BROWN, Charles Talcott	U. of Minnesota	MD 1957	Bellevue Hospital, New York, N. Y.
BROWN, Frank Thomas	U. of Minnesota	MD 1957	Parkland Mem. Hospital, Dallas, Texas
BROWN, Hubert Marion	Johns Hopkins U.	MD 1953	U. of M. Hosp., Dept. Derm., Mpls, Minn.
CADMAN, Norman LeRoy	U. of Chicago	MD 1953	200 1st St. S.W., Rochester, Minn.
CALVERLEY, John Robert	U. of Oregon	MD 1955	200 1st St. S.W., Rochester, Minn.
CASEY, John J.	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
CHAPMAN, George Robert	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis, Minn.
CHRISTENSON, Leland Roger	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
CROW, George Michael	U. of Minnesota	MD 1957	Mercy Hospital, Toledo, Ohio
CUNNINGHAM, Richard Donald	U. of Minnesota	MD 1957	Tripler Army Hospital, Honolulu, Hawaii
DAHL, Diane Anita	U. of Wisconsin	MD 1956	814 Mahtomedi Ave., Mahtomedi, Minn.
DALY, Alfred Emanuel	St. Louis U.	MD 1957	Tripler Army Hospital, Honolulu, Hawaii
DAWSON, Jr., Walter John	U. of Minnesota	MD 1957	St. Luke's Hospital, Cleveland, Ohio
DIVERTIE, Matthew Burgess	U. of Glasgow, Scotland	(Bach. Med. & Surg.) 1947	200 1st St. S.W., Rochester, Minn.
DUDLEY, James Patrick	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
DVORAK, Jr., Benjamin Anthony	Vanderbilt U.	MD 1957	Vanderbilt U. Hospital, Nashville, Tenn.
EHRLICH, Jr., S. Paul	U. of Minnesota	MD 1957	USPHS Hospital, Staten Island, N. Y.
ENGSTROM, E. Duane	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
EUSTERMAN, Joseph Huntimer	U. of Minnesota	MD 1957	Abington Mem. Hospital, Abington, Pa.
FAIRBANKS, Leland L.	U. of Minnesota	MD 1957	USPHS Hospital, New Orleans, La.
FEHR, Peter Eilert	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
FOLEY, Robert Russell	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose, Cal.
GELLER, Joseph	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
GIESBRECHT, Arnold	U. of Manitoba	MD 1957	Hallock, Minn.
GLETNE, John Sanford	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose, Cal.
GOLDFINE, M. Melvin	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
GOLDSTEIN, Alan Lawrence	U. of Minnesota	MD 1957	St. Charles Hospital, Toledo, Ohio
GONIOR, Thomas Henry	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
GOOD, Gary Daniel	U. of Minnesota	MD 1957	Salt Lake Co. Gen. Hosp., Salt Lake City, U.
GOODMAN, Ernest	U. of Bologna, Italy	MD 1954	St. Joseph's Hospital, St. Paul, Minn.
GOODMAN, Malka Lotterstein	U. of Minnesota	MD 1956	1053 Iglehart Ave., St. Paul, Minn.
GRAHAM, Asa Briggs	U. of Minnesota	MD 1957	Mercy Hospital, Toledo, Ohio
HANEY, David George	U. of Minnesota	MD 1957	Wayne Co. Gen. Hosp., Eloise, Mich.
HARDER, Donald S.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
HARTY, Jerome Leo	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
HEGGESTAD, Carl Blixseth	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
HILGER, John Rothrock	McGill U.	MD 1957	Ancker Hospital, St. Paul, Minn.
HILKER, Robert Tolan	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
HOLDCRAFT, John Willard	Jefferson Med. Col.	MD 1956	307 Myrtle Ave., Woodbury, N. J.
HORNS, Norman Mather	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
HOVERSTEN, Vincent Wordsworth	U. of Minnesota	MD 1957	Santa Clara Co. Hospital, San Jose 28, Cal.

Name	*School*		*Address*
JACKISH, George Edward	U. of Minnesota	MD 1957	Hurley Hospital, Flint, Mich.
JAEGER, Dwight E.	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
JOHNSON, Doris I.	U. of Minnesota	MD 1957	Orange Co. Hospital, Orange, Cal.
JOHNSON, Wendell August	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
JOHNSON, William Stark	U. of Illinois	MD 1952	200 1st St. S.W., Rochester, Minn.
KAYUTE, Sheldon Wayne	U. of Minnesota	MD 1949	State Hospital, Willmar, Minn.
KEAIRNES, Harold Wrenn	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
KOZAK, Michael John	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
KVISTBERG, Gerald Kent	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
KYLLONEN, Ronald Rudolph	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
LARSON, Dean Weston	U. of Minnesota	MD 1957	City Hospital, Cleveland, Ohio
LICHTIG, Moses	U. of Minnesota	MD 1957	Montefiore Hospital, New York, N. Y.
LIFSON, William L.	U. of Minnesota	MD 1957	Milw. Co. Hospital, Milwaukee, Wis.
LINDHOLM, Dale David	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
LUND, Richard Roy	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
LYNCH, Richard Patrick	U. of Minnesota	MD 1957	St. Mary's Hospital, Duluth, Minn.
LYON, Fred Alexander	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
MARK, Merle Stanley	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MATSON, Roland Willatz	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.
McGUIRE, Arthur Merritt	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
MENA, Abelardo	National Univ. of South East, Mexico	MD 1952	U. of Minn. Hospitals, Minneapolis, Minn.
MLINAR, Joseph Paul	U. of Minnesota	MD 1957	Phila. Gen. Hospital, Philadelphia, Pa.
MODELL, Jerome Herbert	U. of Minnesota	MD 1957	St. Albans Naval Hosp., St. Albans, L. Is., New York
MOE, Walter Wyatt	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
MORI, Hideo	U. of Chicago	MD 1956	924 Essex St. S.E., Minneapolis, Minn.
MUCHOW, Gene Carleton	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MULVAHILL, John Edward	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
MUSA, Byron Ulysses	U. of Oregon	MD 1956	708 W. Deschutes, Redmond, Ore.
MUZZALL, Hugh Arthur	U. of Wash., Seattle	MD 1956	206 E. 10th, Ellensburg, Wash.
NADEAU, Pierre	U. of Montreal	MD 1954	200 1st St. S.W., Rochester, Minn.
NAROS, Jack Albert	U. of Minnesota	MD 1957	So. Pacific Gen. Hosp., San Francisco, Cal.
NIAZI, Suad Asad	Royal Col. Med., Baghdad, Iraq	Bach. Med. & Surg. 1941	Box 446, Clarkfield, Minn.
NIELSEN, Andrew Adolf	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
NILSEN, John Anton	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
OLSON, Robert Joseph	U. of Minnesota	MD 1957	USPHS Hospital, San Francisco, Cal.
OPHEIM, Richard Henry	U. of Minnesota	MD 1957	So. Pacific Gen. Hosp., San Francisco, Cal.
ORBUCH, Martin Walter	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
OSE, Anna Leonija	U. of Latvia, Riga, Latvia	"Phys." 1931	State School & Hospital, Cambridge, Minn.
OSS, Ausma Velta	U. of Minnesota	MD 1957	St. Michael Hospital, Newark, N. J.
PAWLYSZYN, Julian	U. of Heidelberg, Germany	MD 1949	State Hospital, Anoka, Minn.
PETERSON, Charles Allan	U. of Minnesota	MD 1957	USPHS Hospital, Boston, Mass.
PETERSON, John Alfred	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
POBOR, Paul H.	U. of Minnesota	MD 1957	Los Angeles Co. Hosp., Los Angeles, Cal.
POLLARD, John William	U. of Minnesota	MD 1957	Valley Forge Army Hosp., Phoenixville, Pa.
REEMSNYDER, Curtis C.	U. of Minnesota	MD 1957	Bethesda Hospital, St. Paul, Minn.
ROACH, Charles Albert	U. of Minnesota	MD 1957	Miller Hospital, St. Paul, Minn.
ROBINSON, James Parker	U. of Minnesota	MD 1957	Walter Reed Army Hospital, Wash., D. C.
RUSHAY, Arthur James	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
RYDBERG, John S.	U. of Minnesota	MD 1957	Receiving Hospital, Detroit, Mich.
SACKS, Jerome Henry	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
SAYTHER, Keith Duddley	U. of Minnesota	MD 1957	San Joaquin Co. Hosp., French Camp, Cal.
SCHEREK, Jerome John	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SCHLOFF, Ivan	U. of Bologna, Italy	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
SCHROEPFER, Jr., George John	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.
SHAPIRO, Stanley William	U. of Minnesota	MD 1957	Mt. Sinai Hospital, Minneapolis, Minn.
SHEFVELAND, John Ronald	U. of Minnesota	MD 1957	Harbor Gen. Hospital, Torrance, Cal.
SHUMRICK, Donald Albert	U. of Minnesota	MD 1957	San Fran. Cty.-Co. Hosp., San Fran., Cal.
SINGBEIL, Julius Henry	U. of Manitoba	MD 1956	123 N. 6th St., Breckenridge, Minn.
SMITH, Henry Nippert	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SOLLIE, Stanley Carol	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
SPRAITZ, Jr., Anton Francis	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
SUTHER, Lennart Elmer	U. of Minnesota	MD 1957	Highland-Alameda Co. Hosp., Oakland, Cal.
TANQUIST, Jr., Edwin John	U. of Minnesota	MD 1957	Gorgas Hospital, Ancon, Canal Zone
THEOBALT, Inge Marie Bartsch	U. of Kiel, Germany	MD 1944	U. of Minn. Hospitals, Minneapolis, Minn.
THOMPSON, Wayne William	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
TOUSSAINT, Florence Amalia	U. of Minnesota	MD 1957	Queen's Hospital, Honolulu, Hawaii
VALGEMAE, Romil	U. of Minnesota	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
VAN DE WATER, Frank W.	U. of Minnesota	MD 1957	Denver City Gen. Hosp., Denver, Colo.
VAN PUFFELEN, Paul S.	U. of Minnesota	MD 1957	St. Luke's Hospital, Duluth, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Name	*School*		*Address*
WELLS, Ralph Frederick	U. of Minnesota	MD 1957	Brooke Army Hospital, San Antonio, Tex.
WILKEN, Robert Wayne	U. of Minnesota	MD 1957	Ancker Hospital, St. Paul, Minn.
WILSON, George Patterson	Washington U., Mo.	MD 1956	Vet. Adm. Hospital, Minneapolis, Minn.
WORLTON, Jr., James Timbrell	U. of Utah	MD 1954	200 1st St. S.W., Rochester, Minn.
YOUNG, Joseph Orville	U. of Minnesota	MD 1957	Tripler Army Hospital, Honolulu, Hawaii
ZAK, Solomon Joseph	U. of Minnesota	MD 1957	U. of Minn. Hospitals, Minneapolis, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
CHAPPELL, Elliott Rae	U. of Nebraska	MD 1944	112 S. Main St., Stillwater, Minn.
FALK, Abraham	Albany Med. Col.	MD 1936	2500 Como Ave., St. Paul, Minn.
FOX, Philip Martin	U. of California	MD 1947	Vet. Adm. Hospital, Minneapolis, Minn.
GERBER, Edward Paul	U. of Illinois	MD 1953	200 1st St. S.W., Rochester, Minn.
GIDDINGS, Frank Chandler	U. of Nebraska	MD 1954	Miller Hospital, St. Paul, Minn.
HUFF, James Foster	Louisiana State U.	MD 1955	200 1st St. S.W., Rochester, Minn.
LINKÉ, Clara Julia Leinberger	U. of Illinois	MD 1950	St. Luke's Hospital, Duluth, Minn.
McCARTHY, John Denis	U. of Rochester	MD 1955	200 1st St. S.W., Rochester, Minn.
McHUTCHISON, Samuel Kerr	U. of Pennsylvania	MD 1955	St. Charles, Minn.
SCHNABEL, Robert Frederick	Med. Col. of Va.	MD 1951	220 S. Broadway, Crookston, Minn.
WARDEN, Herbert Edgar	U. of Chicago	MD 1946	U. of Minn. Hosp., Dept. Surg., Mpls.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
ABSOLON, Karel Bedrich	Yale U.	MD 1952	Key West Naval Hosp., Key West, Fla.
ANDERSEN, James Gordon	Harvard U.	MD 1952	U. S. Vet. Adm. Hosp., Boise, Idaho
GIULIANI, Emilio R.	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
HAWK, Dale Jay	Hahnemann, Pa.	MD 1951	St. Charles, Minn.
JEWELL, Jr., Albert Hartwell	Northwestern U.	MD 1950	209 W. Mill St., Austin, Minn.
STEIDL, Richard Meredith	Albany Med. Col.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
TORO-NAZARIO, Rafael Andres	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
VASTOLA, Jr., Anthony Paul	Boston U.	MD 1953	200 1st St. S.W., Rochester, Minn.
VOGEL, Gerald William	U. of Chicago	MD 1954	200 1st St. S.W., Rochester, Minn.
WAGNER, Robert Morris	Cornell U.	MD 1952	507 Med. Arts Bldg., Minneapolis, Minn.

PHYSICIANS LICENSED NOVEMBER 8, 1957

October, 1957, Examination

Name	*School*		*Address*
CHEDISTER, Charles Russell	U. of Illinois	MD 1956	4119 2nd Ave. So., Minneapolis, Minn.
CONN, Jr., Rex Boland	Yale Univ.	MD 1953	U. of Minn. Hospitals, Minneapolis, Minn.
DEASON, Keith Burdette	Creighton U.	MD 1957	St. Luke's Hospital, Duluth, Minn.
DIRE, William Nick	U. of Oregon	MD 1957	Mpls. Gen. Hospital, Minneapolis 15, Minn.
ECKHARDT, Arthur Lloyd	U. of Oregon	MD 1957	Ancker Hospital, St. Paul, Minn.
EICHENHOLZ, Alfred	Ludwig-Maximilian U., Munich, Germany	MD 1951	Vet. Adm. Hospital, Minneapolis, Minn.
FLOYD, Malcolm Stafford	Med. Col. of S. Car.	MD 1956	200 1st St. S.W., Rochester, Minn.
FLUEGEL, John Ormond	Creighton U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
FORBES, Edward Francis	Creighton U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
GILBERT, Robert Alfred	Columbia U.	MD 1956	200 1st St. S.W., Rochester, Minn.
HAKALA, Edwin Wayne	U. of Minnesota	MD 1938	505 Beltzell Ave., Fort Benning, Ga.
JENKINS, Wallace Vernon	U. of Utah	MD 1955	Mpls. Gen. Hospital, Minneapolis 15, Minn.
JOHNSON, Thomas Errol	U. of Nebraska	MD 1954	Miller Hospital, St. Paul, Minn.
KNUDSEN, Jerry Milo	Baylor Univ.	MD 1956	Ancker Hospital, St. Paul, Minn.
LINDSETH, Esten Opland	U. of Oslo, Norway	MD 1949	U. of M. Grad School, Minneapolis, Minn.
McQUARRIE, Donald Gray	U. of Utah	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
O'LOUGHLIN, Peter Daniel	Marquette U.	MD 1955	St. Mary's Hospital, San Francisco, Cal.
RAKOW, Raymond William	U. of California	MD 1953	200 1st St. S.W., Rochester, Minn.
RECHT, Thomas Monroe	U. of Nebraska	MD 1956	Mpls. Gen. Hospital, Minneapolis 15, Minn.
REISCH, Alvin J.	Northwestern U.	MD 1956	200 1st St. S.W., Rochester, Minn.
REYNOLDS, James Francis	U. of Penna.	MD 1956	200 1st St. S.W., Rochester, Minn.
REYNOLDS, William Arthur	Washington U. (Mo.)	MD 1956	200 1st St. S.W., Rochester, Minn.
SCHORR, William Francis	Marquette U.	MD 1957	St. Mary's Hospital, Duluth, Minn.
SEIFERT, Richard Henry	Loyola Univ.	MD 1957	St. Joseph's Hospital, St. Paul, Minn.
SHANAHAN, Daniel Francis	Creighton U.	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
WHITMAN, Edwin Joel	U. of Pittsburgh	MD 1956	200 1st St. S.W., Rochester, Minn.

RECIPROCITY CANDIDATES

Name	*School*		*Address*
AHERN, Gene	U. of Illinois	MD 1953	1218 S. Park, Red Wing, Minn.
BALLANTINE, Jerome Jennings	Indiana Univ.	MD 1956	Vet. Adm. Hospital, Minneapolis, Minn.
BOLLIGER, Eugene Frederick	U. of Michigan	MD 1946	Northwestern Hospital, Minneapolis, Minn.
BRODHUN, John Charles	Marquette U.	MD 1954	915 3rd Ave. S.E., Rochester, Minn.
BROWN, Paul Marvin	Harvard U.	MD 1949	200 1st St. S.W., Rochester, Minn.

Name	*School*		*Address*
BYNUM, Jr., Grover LaFayette	Tulane U.	MD 1953	200 1st St. S.W., Rochester, Minn.
CARVETH, Stephen Walker	U. of Nebraska	MD 1956	200 1st St. S.W., Rochester, Minn.
COWGER, Marilyn Louise	U. of Nebraska	MD 1956	200 1st St. S.W., Rochester, Minn.
CULLEN, Jr., Paul Kent	U. of Indiana	MD 1956	200 1st St. S.W., Rochester, Minn.
DAW, Edward Francis	State U. of Iowa	MD 1954	200 1st St. S.W., Rochester, Minn.
DOMBROWSKI, Jr., Edmund T.	U. of California	MD 1956	200 1st St. S.W., Rochester, Minn.
GILDERSLEEVE, Robert G.	Col. Med. Evang.	MD 1948	Watertown, Minn.
GILLOON, James Robert	Creighton U.	MD 1953	200 1st St. S.W., Rochester, Minn.
GRAUDINS, Gunars	U. of Iowa	MD 1956	200 1st St. S.W., Rochester, Minn.
HEILMAN, Richard Owen	U. of Wisconsin	MD 1955	1003 Oak St., Tomah, Wis.
HENKE, Frederick William	U. of Wisconsin	MD 1945	Vet. Adm. Hospital, Minneapolis, Minn.
INLOW, Robert Pierson	Indiana U.	MD 1956	200 1st St. S.W., Rochester, Minn.
KEIG, Jr., William Paul	Northwestern U.	MD 1947	1600 University Ave., Grand Forks, N. D.
KINNARD, Jr., John Parkes	U. of Tennessee	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
KUNDEL, Robert Ray	U. of Iowa	MD 1956	208 E. Chapman, Ely, Minn.
LILLIE, Andrew Rendall	U. of Manitoba	MD 1956	U. of Minn. Hospitals, Minneapolis, Minn.
LORENC, Ernest	U. of Iowa	MD 1954	200 1st St. S.W., Rochester, Minn.
McPARLAND, Jr., Felix Augustus	Western Reserve	MD 1950	4841 E. Lake Harriet Blvd., Mpls., Minn.
MILLER, Jr., Archie William	U. of Michigan	MD 1956	200 1st St. S.W., Rochester, Minn.
MUNGER, James Everett	Ohio State U.	MD 1954	8506 Beverly St., Duluth 8, Minn
NELSON, Donald Keith	Indiana U.	MD 1956	200 1st St. S.W., Rochester, Minn.
NORBECK, David Eskel	U. of Illinois	MD 1952	303 Doctors Bldg., Minneapolis, Minn.
POSEY, John William	U. of Nebraska	MD 1951	200 1st St. S.W., Rochester, Minn.
SCHEEN, Jr., Sanuel Randolph	U. of Louisville	MD 1953	200 1st St. S.W., Rochester, Minn.
STORY, Jimmy Lewis	Vanderbilt U.	MD 1955	U. of Minn. Hospitals, Minneapolis, Minn.
TAYLOR, Lawrence Carol	U. of Nebraska	MD 1955	200 1st St. S.W., Rochester, Minn.
TIPPITT, Nathaniel George	Louisiana State U.	MD 1950	200 1st St. S.W., Rochester, Minn.
WILEY, Alden Francis	U. of Iowa	MD 1953	Box 176, Kalkaska, Mich.

NATIONAL BOARD CANDIDATES

Name	*School*		*Address*
ADAMS, Ralph Edwin	U. of Oregon	MD 1956	200 1st St. S.W., Rochester, Minn.
ALLAMAN, Loren Ellsworth	Northwestern U.	MD 1954	200 1st St. S.W., Rochester, Minn.
BATTAILE, William George	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.
BERNSTEIN, Eugene Felix	State U. of N. Y.	MD 1954	U. of Minn. Hospitals, Minneapolis, Minn.
BOROSON, Hugh Norbert	New York Univ.	MD 1951	Northw'n Hosp., Thief River Falls, Minn.
BRINK, Benno Marcellus	Creighton U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CLARK, Lealand L.	Columbia U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CORR, William Philip	Stanford U.	MD 1956	200 1st St. S.W., Rochester, Minn.
CRANDALL, Earle Ellsworth	U. of Illinois	MD 1956	200 1st St. S.W., Rochester, Minn.
DAVIDSON, Paul	Albany Med. Col.	MD 1956	200 1st St. S.W., Rochester, Minn.
DONDERO, Jr., John A.	Georgetown U.	MD 1952	58 85th St., Brooklyn 9, N. Y.
EDELSTEIN, Jack Paul	U. of Chicago	MD 1956	200 1st St. S.W., Rochester, Minn.
ENGLER, Robert Sherman	Columbia U.	MD 1954	200 1st St. S.W., Rochester, Minn.
ETZWILER, Donnell Dencil	Yale Univ.	MD 1953	445 E. 68th St., New York 21, N. Y.
FARLEY, Harrison Hatheway	Cornell Med. Col.	MD 1954	Mpls. Gen. Hospital, Minneapolis 15, Minn.
FAUST, Herbert Arlington	Hahnemann Med. Col.	MD 1952	200 1st St. S.W., Rochester, Minn.
GRABOW, Jack David	U. of Buffalo	MD 1956	200 1st St. S.W., Rochester, Minn.
HIGGINS, John Elton	Dalhousie U.	MD 1951	200 1st St. S.W., Rochester, Minn.
HOOPS, Jr., Harold John	Columbia U.	MD 1954	200 1st St. S.W., Rochester, Minn.
HOPKINS, Donald Marks	Northwestern U.	MD 1956	200 1st St. S.W., Rochester, Minn.
HUNT, James Cleon	U. of Illinois	MD 1954	200 1st St. S.W., Rochester, Minn.
KERN, Kathryn Eustance	U. of Rochester	MD 1956	4921 Miss. Court, Minneapolis, Minn.
KERN, Jr., William Albert	U. of Rochester	MD 1956	Mpls. Gen. Hospital, Minneapolis 15, Minn.
KINZEL, Raymond Christian	New York Med. Col.	MD 1955	200 1st St. S.W., Rochester, Minn.
KIRSHEN, Robert	Chicago Med. Sch.	MD 1956	200 1st St. S.W., Rochester, Minn.
MAYTUM, Wellington James	U. of Penna.	MD 1956	200 1st St. S.W., Rochester, Minn.
MELNICK, Gilbert Stanley	State U. of N. Y.	MD 1954	1338 W. Maynard Dr., St. Paul, Minn.
MURPHY, Frank Patrick	Loyola U.	MD 1954	200 1st St. S.W., Rochester, Minn.
NYDELL, Jr., Carl Clifford	Col. Med. Evang.	MD 1954	200 1st St. S.W., Rochester, Minn.
REX, James Caldwell	Temple U.	MD 1950	200 1st St. S.W., Rochester, Minn.
RICE, Bernard Francis	Temple U.	MD 1956	200 1st St. S.W., Rochester, Minn.
SHERRICK, Donald William	Yale Univ.	MD 1956	200 1st St. S.W., Rochester, Minn.
SPIEKERMAN, Ralph Earl	U. of Illinois	MD 1954	200 1st St. S.W., Rochester, Minn.
SPURGEON, Frederick Calvin	New York Med. Col.	MD 1953	200 1st St. S.W., Rochester, Minn.
THOMPSON, III, John William	St. Louis Univ.	MD 1954	200 1st St. S.W., Rochester, Minn.
WEAVER, Walt Franklin	Western Reserve	MD 1956	200 1st St. S.W., Rochester, Minn.
WEBB, Harry Edward	U. of Kansas	MD 1956	200 1st St. S.W., Rochester, Minn.
WESTLIN, Jr., William Frederick	New York Med. Col.	MD 1954	200 1st St. S.W., Rochester, Minn.
WHERRY, David Colwell	Geo. Washington U.	MD 1952	504 Monticello Blvd., Alexandria, Va.
WHITE, James Colin	U. of Vermont	MD 1956	200 1st St. S.W., Rochester, Minn.
WOELLNER, Richard Carlton	U. of Chicago	MD 1955	Sch. Aviation Med., Naval Air Station, Pensacola, Fla.
WRY, Paul Edward	Georgetown U.	MD 1953	200 1st St. S.W., Rochester, Minn.

Meetings and Announcements

STATE

MINNESOTA STATE MEDICAL ASSOCIATION, 105th annual meeting, Minneapolis, May 22, 23 and 24, 1958. Business sessions and exhibits, Minneapolis Auditorium. Headquarters, Leamington Hotel.

NATIONAL

American College of Surgeons, sectional meeting, Des Moines, Iowa, March 27-29, 1958.

American Congress of Physical Medicine and Rehabbilitation, 36th annual scientific and clinical session, Bellevue-Stratford Hotel, Philadelphia, August 24-29, 1958.

American Gastroenterological Association, 59th annual meeting, Washington, D. C., May 30-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

Chicago Medical Society, Annual Clinical Conference, Palmer House, Chicago, Illinois, March 4, 5, 6, and 7, 1958.

INTERNATIONAL

Fifth International Congress on Diseases of the Chest, sponsored by American College of Chest Physicians, Tokyo, Japan, September 7-11, 1958.

Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

International College of Surgeons, 11th biennial International Congress, in conjunction with 23rd annual Congress of United States and Canadian Sections (North American Federation), Los Angeles, California, March 9-14, 1958.

International Society of Internal Medicine, Fifth International Congress of Internal Medicine, Philadelphia, Pennsylvania, April 24-26, 1958. Edward R. Loveland, Secretary-General, 4200 Pine Street, Philadelphia 4, Pennsylvania.

Third International Congress of Allergy, sponsored by International Association of Allergology and French Allergy Association, Paris, France, October 19-26, 1958.

World Congress of Gastroenterology, Washington, D. C., May 25-31, 1958. H. M. Pollard, M.D., Secretary-General, University Hospital, Ann Arbor, Michigan.

ISAAC RAY LECTURE SERIES

Dr. Manfred S. Guttmacher, psychiatrist to the Supreme Bench of Baltimore, will speak Monday, Tuesday and Thursday evenings, March 3, 4 and 6, in the 1958 Isaac Ray lecture series to be at the University of Minnesota under the joint sponsorship of the University's law and medical schools.

Dr. Guttmacher will discuss "Confidentiality and Privileged Communication," "The Psychotic Murderer" and "Psychiatric Court Clinics" in his three lectures which will be presented primarily for doctors and lawyers. The lectures, which will be given in Mayo Auditorium, will begin at 8 p.m. each of the three evenings.

POSTGRADUATE COURSE

The second Annual Postgraduate Course in Fractures and Other Trauma will be given by the Chicago Committee on Trauma of the American College of Surgeons, for four days from Wednesday, April 16, through Saturday, April 19, at the John B. Murphy Memorial Auditorium, 40 East Erie Street, Chicago, Illinois.

SECTIONAL SURGICAL MEETING IN DES MOINES, IOWA

All members of the medical profession are invited to attend a three-day Sectional Meeting of the American College of Surgeons in Des Moines, Iowa, March 27 through 29, at the Hotel Fort Des Moines.

Topics will include emergency care of multiple injuries, surgery for congenital lesions, cardiac arrest, cancer, jaundiced patient, ovarian tumors, fluids and electrolytes. Medical motion pictures will also be shown daily, with an especially selected program scheduled for Thursday evening.

GENERAL MEDICINE AND SURGERY LECTURES

A three-day program of lectures and discussions on problems of current interest in general medicine and surgery will be presented by staff members of the Mayo Clinic and the Mayo Foundation for Medical Education and Research, April 14, 15 and 16, 1958. Up to twenty-one hours of Category I credit may be obtained by American Academy of General Practice members who attend. There are no fees for this program.

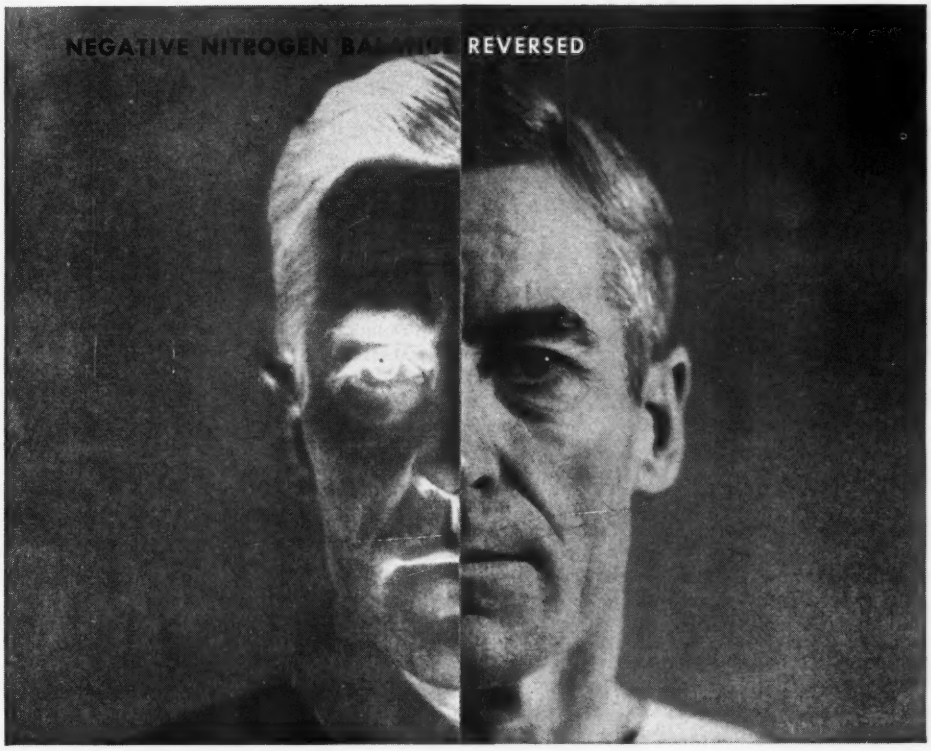
CONTINUATION COURSES

Medical continuation courses to be presented at the Center for Continuation Study, University of Minnesota, are as follows:

March 3-5.....Pediatrics for General Physicians
March 17-19.....Internal Medicine for Internists
March 20-22.....Surgery for Surgeons
April 7-9.....Radiology for General Physicians

For further information concerning the above courses, write to the Director, Department of Continuation Medical Education, 1342 Mayo Memorial, University of Minnesota, Minneapolis 14.

(Continued on Page A-42)



Nilevar®

stimulates protein synthesis, corrects negative nitrogen balance

Increased nitrogen loss, with resulting negative nitrogen balance, occurs in infection, trauma, major surgery, extensive burns, certain endocrine disorders and starvation and emaciation syndromes. The intrinsic control of protein metabolism is lost and a protein "catabolic state" occurs. A patient requiring more than ten days of bedrest usually has had sufficient metabolic insult[1] to precipitate such a "catabolic" phase.

Nilevar (brand of norethandrolone) has been used in patients with varied conditions including hyperthyroidism, poliomyelitis, aplastic anemia, glomerulonephritis, anorexia nervosa and postoperative protein depletion. The patients gained weight and felt better. It was concluded[2] that "the drug certainly caused a reversal of rather recalcitrant or progressive catabolic patterns of disease."

Nilevar is unique among anabolic steroids in that androgenic side action is minimal or absent.

The suggested adult dosage is three to five tablets (30 to 50 mg.) daily. For children 1.5 mg. per kilogram of weight is recommended.

G. D. Searle & Co., Chicago 80, Illinois. Research in the Service of Medicine.

---

1. Axelrod, A. E.; Beaton, J. R.; Cannon, P. R., and others: Symposium on Protein Metabolism, New York, The National Vitamin Foundation, Incorporated, (March) 1954, p. 100.

2. Proceedings of a Conference on the Clinical Use of Anabolic Agents, Chicago, Illinois, G. D. Searle & Co., April 9, 1956, pp. 32-35.

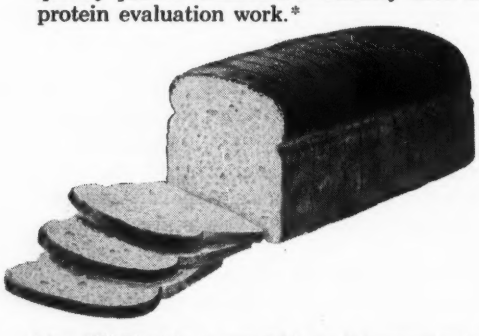
SEARLE

V^{10} protein is a COMPLETE PROTEIN

Complete protein is essential to the maintenance of body cells

Constant daily replacement of protein forming the cells of blood, skin, muscle, nerve, bone, and even teeth, is necessary to maintain health and vigor. In order that this process be maintained, the diet must contain adequate quantities of "complete protein" with all of the essential amino acids for simultaneous ingestion.

The Wisconsin Alumni Research Foundation has licensed the production of such a complete protein in the form of V^{10} Protein Concentrate. V^{10} Protein is composed entirely of grains, yet results of laboratory tests by the Foundation show that it has a protein efficiency value equal to casein, the high quality protein standard commonly used in protein evaluation work.*



Now V^{10} Protein is available in Minnesota in V^{10} Protein Bread. This light, delicious bread adds variety to the daily dietary requirement for protein. V^{10} Protein Bread will greatly aid in the planning of meals and will help promote health and vigor for all age groups.

WISCONSIN ALUMNI RESEARCH FOUNDATION

*A complete report on these animal feeding studies is available on request. Address WISCONSIN ALUMNI RESEARCH FOUNDATION, P. O. Box 2217, Madison 1, Wis.

(Continued from Page 146)

THREE REGIONAL INSTITUTES ON PRINCIPLES OF EPIDEMIOLOGY

Three Regional Institutes on Principles of Epidemiology will be sponsored by the Minnesota Department of Health and University of Minnesota, School of Public Health, School of Veterinary Medicine in co-operation with the U. S. Department of Health, Education and Welfare, Public Health Service, Bureau of State Service, Communicable Disease Center Training Branch.

Dates and locations for the institute are as follows:

Fergus Falls.....February 25, 26, 27, 28, 1958
Duluth.....March 3, 4, 5, 6, 1958
Mankato.....March 4, 5, 6, 7, 1958

The course is designed for health officers, physicians, nurses, veterinarians, laboratory workers, environmental health personnel and other members of the public health.

For additional information, write to D. S. Fleming, M.D., Director, Division of Disease Prevention and Control, Minnesota Department of Health, University Campus, Minneapolis 14, Minnesota.

COMMITTEE ACTION

Child Health Committee

(Continued from Page 139)

2. *Promotional*

   (a) Make surveys to ascertain what children are not now receiving continuous health supervision

   (b) Cooperate with other agencies in promoting the planned program of periodic health appraisal most suitable to any given community at the time of study

   (c) Assist in developing school health councils or subcouncils on school health under already existing community councils

   (d) Irrespective of what special medical examinations a child may need, recommend that primary general examination be made by the family physician or pediatrician

   (e) Aid in development of cumulative child health records and their use by parents, physicians, dentists, and schools

   (f) Promote research in child health programs as cooperative activities involving medicine, education, and public health

The Committee on Child Health of the Minnesota State Medical Association takes this opportunity to endorse these recommendations.

ELDON BERGLUND, M.D.
Liaison Representative to the PTA Committee on Child Health

when are tranquilizers indicated in pediatrics ?

Some doctors have questioned the use of tranquilizers in children. They feel, and rightly so, that these drugs should not be used as palliatives to mask distressing symptoms, while etiological factors go uncorrected. But there are three situations in which even the most conservative physician would not hesitate to use tranquilizers:

1. When the usually well-adjusted child needs a buffer against temporary emotional stress, such as hospitalization.
2. When a child needs relief from an anxiety-reaction that is in turn anxiety-provoking, so as to pave the way for basic therapy.
3. When anxiety underlies or complicates somatic disease, as in asthma.

In such situations, tranquilizers are likely to be more effective and better tolerated than previously accepted therapy, such as barbiturates.

But the question arises: **which** tranquilizer is suitable for children?

Most of the physicians now using tranquilizers in pediatric practice have found the answer to be ATARAX, confirming the conclusions of repeated clinical studies.

ATARAX is effective in a wide range of pediatric indications.
ATARAX has produced a "striking response" in a wide range of hyperemotive states.* In a study of 126 children, "the calming effect of hydroxyzine (ATARAX) was remarkable" in 90%.* Among the conditions that are improved with ATARAX are tics, nervous vomiting, stuttering, temper tantrums, disciplinary problems, crying spasms, nightmares, incontinence, hyperkinesia, etc.*

ATARAX is well tolerated even by children.
"ATARAX appears to be the safest of the mild tranquilizers. Troublesome side effects have not been reported. . . ."*

ATARAX offers two pediatric dosage forms.
ATARAX Syrup is especially designed for acceptability by medicine-shy youngsters. A small 10 mg. tablet is also available. In either case, you will get a rapid, uncomplicated response. Why not, for the next four weeks, prescribe ATARAX for your hyperemotive pediatric patients. See whether you, too, don't find it eminently suitable.

*Documentation on request

PEACE OF MIND ATARAX®
(BRAND OF HYDROXYZINE)

ATARAX
in any hyperemotive state

for childhood behavior disorders
10 mg. tablets—3-6 years, one tablet t.i.d.; over 6 years, two tablets t.i.d. Syrup—3-6 years, one tsp. t.i.d.; over 6 years, two tsp. t.i.d.

for adult tension and anxiety
25 mg. tablets—one tablet q.i.d. Syrup—one tbsp. q.i.d.

for severe emotional disturbances
100 mg. tablets—one tablet t.i.d.

for adult psychiatric and emotional emergencies
Parenteral Solution—25-50 mg. (1-2 cc.) intramuscularly, 3-4 times daily, at 4-hour intervals. Dosage for children under 12 not established.

Supplied: Tablets, bottles of 100. Syrup, pint bottles. Parenteral Solution, 10 cc. multiple-dose vials.

M. A. Seidell M.D.
Medical Director

Rx ROERIG
New York 17, New York
Division, Chas. Pfizer & Co., Inc.

EVERY WOMAN

WHO SUFFERS

IN THE

MENOPAUSE

DESERVES

"PREMARIN"

widely used

natural, oral

estrogen

AYERST LABORATORIES

New York, N.Y. • Montreal, Canada

5646

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building, Saint Paul 1, Minnesota

F. H. Magney, M.D., Secretary

Minneapolis Nurse Sentenced On Basic Science Charge

On December 2, 1957, a registered nurse, Mrs. Lois Kieser, twenty-six years of age, 5608 Osseo Road, Minneapolis, was sentenced by the Hon. Harold N. Rogers, Judge of the District Court of Hennepin County, to a term of one year in the Hennepin County jail. However, the Court then stayed the execution of the sentence for a period of one year and placed the defendant under the supervision of the Hennepin County Probation Office during this time. Judge Rogers imposed two conditions in connection with the stay of execution: (1) that the defendant refrain from the personal use of narcotic drugs except under the direction and prescription of a physician and, (2) that she consult a doctor of medicine once each month in reference to her health and authorize him to make a monthly report to the probation office. Mrs. Kieser was originally charged in this case with a felony, obtaining narcotic drugs by the forgery of a prescription. However, the charge was later reduced by the Court to practicing healing without having a certificate of registration in the basic sciences and Mrs. Kieser entered a plea of guilty before Judge Rogers to the reduced charge on September 20, 1957.

The defendant was arrested on September 17, 1957, after a Robbinsdale, Minnesota, pharmacist reported that he had delivered a 30 cc. vial of demerol to a woman at the Kieser home and received in exchange for the demerol a narcotic prescription purportedly signed by a Minneapolis doctor of medicine. When the physician was later contacted, he stated that the entire prescription was a forgery. A criminal complaint charging the defendant with the commission of a felony, as stated above, was then signed by a representative of the Minnesota State Board of Medical Examiners, the complaint being issued by the office of the Hennepin County attorney. When Mrs. Kieser was arrested she admitted forging not only the prescription in question but also five or six other prescriptions for demerol. In a signed statement, Mrs. Kieser said that she was born in Rock Elm Township, Wisconsin, on October 25, 1931, and is a registered nurse in the State of Minnesota. According to her statement, the defendant first began the personal use of demerol in February, 1957, and obtained some of the drug in a Minneapolis hospital where she was employed, by diverting to her own use a portion of the demerol that had been ordered for patients.

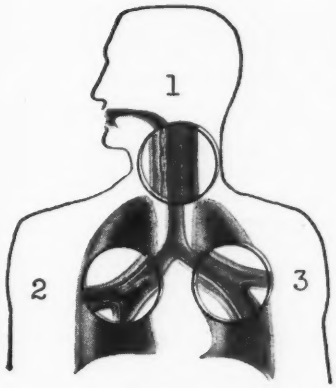
RADIUM RENTAL SERVICE

4340 CEDARWOOD ROAD
MINNEAPOLIS 16, MINNESOTA
TEL. FE 3-5297

Radium element prepared in type of applicator requested

ORDER BY TELEPHONE OR MAIL
PRICES ON REQUEST

breaks up cough*



*Drawing shows how 3-pronged attack of Pyribenzamine Expectorant with Ephedrine breaks up cough by: (1) reducing histamine-induced congestion and irritation throughout the respiratory tract; (2) liquefying thick and tenacious mucus; (3) relaxing bronchioles. Pyribenzamine Expectorant with Codeine and Ephedrine also available (exempt narcotic). Pyribenzamine® citrate (tripelennamine citrate CIBA). **C I B A**

Woman's Auxiliary

ZUMBRO VALLEY AUXILIARY MEETS

The members of the Zumbro Valley Medical Auxiliary met at the Mayo Foundation House January 8 at 2:30 p.m.

Tea was served, followed by a business meeting.

Dr. Frank Krusen, head of the Mayo Clinic Section of Physical Medicine and Rehabilitation, spoke on the progress of the Ability Building Center in Rochester. Mr. David Griggs, Director of the Center, also reported on the activities of the past year.

RAMSEY COUNTY AUXILIARY NEWS

The Ramsey County Auxiliary resumed its activities after the holiday season with a board meeting on January 13 and a luncheon a week later in the Minnesota Club. The two tuberculosis essay winners, students at St. Agnes High School, attended and were presented with cash awards contributed by the Auxiliary and the Ramsey County Tuberculosis and Health Association. Three students from the American Field Services were introduced by Mrs. M. M. Sarnecki, who is affiliated with the service in St. Paul. Two were natives of the Philippines and Iceland, and the third, David Nelson, Minneapolis, of St. Thomas College, spent the summer in Denmark. These young people gave interesting accounts of their experiences. The Mmes. H. O. Peterson, Herman Wolff, and O. I. Sohlberg, the committee in charge of the AAPS essay contest, reported that they are putting notices in the high schools to stimulate interest in the contest.

Mrs. C. L. Oppegaard, state president, was honor guest at the luncheon. The president greeted the members, urging everyone to attend the Mid-Winter Board meeting in the Minnesota Club. Tribute was paid to the memory of a former president, Mrs. A. G. Schulze, who died recently. Mrs. Schulze served very ably from 1927-29. The Auxiliary accepted the board's recommendation to send a bequest in memory of Mrs. Harold Wahlquist, to the State Auxiliary to be used as the state committee sees fit. It was also decided to give a grant of $100 to KTCA for the purpose of furthering educational TV programs.

SEASON'S GREETINGS FROM FORMOSA

"The red color of the card upon which this greeting is written is the favorite color for festivals, and firecrackers are in vogue for every occasion. Our New Year is celebrated but their 'New Year' is really celebrated. It will begin February 18 and then it's a continuous feasting and everyone goes in debt again till the next year's end, when checks are issued to pay the bills (many rubber ones)!! So it's a continuous 'being in the red!!!'

"It seems there is a story with every patient—each one knows that he cannot pay for the medicine used, so when a live chicken is brought to the door we know, too, that the family is offering a real sacrifice in appreciation for health.

"We think of all of our dear friends very often and we wish you all the joys of the season."

THEO and P. J. PANKRATZ

(Text of a greeting to the auxiliary from Mrs. Pankratz to Mrs. A. B. Rosenfield.)

MARGARET SHEMAN WAHLQUIST MEMORIAL COMMITTEE NAMED

A committee to consider an appropriate memorial in tribute to Mrs. Harold Wahlquist, state and national auxiliary leader, who died August 9, 1957, has been named by Mrs. C. L. Oppegaard, state auxiliary president.

Memorial possibilities suggested to date include contributions to cancer research or to the American Medical Education Foundation Fund. A memorial trust fund for scholarships bearing the name of Mrs. Wahlquist has also been suggested. Contributions will be received on a voluntary basis.

Members of the committee are: Mrs. L. P. Howell, Rochester; Mrs. H. H. Fesler, St. Paul; Mrs. Harold Benjamin, Minneapolis; Mrs. Peter Rudie, Duluth; and Mrs. O. M. Heiberg, Worthington.

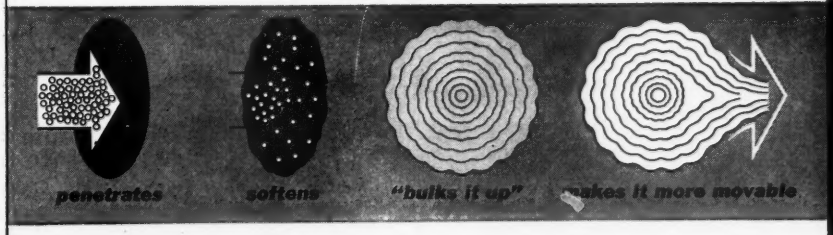
a *penetrant* emulsion for chronic constipation

KONDREMUL® *(PLAIN)*

COLLOIDAL EMULSION OF MINERAL OIL AND IRISH MOSS

permeates the hard, stubborn stool of chronic constipation with millions of microscopic oil droplets, each encased in a film of Irish moss... makes it more movable

KONDREMUL / PATCH



KONDREMUL *(Plain)*—Pleasant-tasting and non-habit-forming. Contains 55% mineral oil. Supplied in bottles of 1 pt.

KONDREMUL *(With Cascara)*—0.66 Gm. nonbitter Ext. Cascara per tablespoon. Bottles of 14 fl.oz.

KONDREMUL *(With Phenolphthalein)*—0.13 Gm. phenolphthalein (2.2 gr.) per tablespoon. Bottles of 1 pt.

When taken as directed before retiring, KONDREMUL does not interfere with absorption of essential nutrients.

THE E. L. PATCH CO. — STONEHAM, MASSACHUSETTS

In Memoriam

MARTIN AUNE

Dr. Martin Aune, eighty-four, died January 4, 1958. A practicing Minneapolis physician for fifty years, Dr. Aune was a member of Hennepin County Medical Society, Theta Kappa Psi medical fraternity, Minneapolis Lions Club, Minnesota Chapter of National Sojourners, Khurum Masonic Lodge 112, Zuhrah Temple and Scottish. He was also a life member of the Minnesota State Medical Association.

Dr. Aune, lieutenant colonel on the general staff of the Minnesota National Guard, and a World War I veteran, was appointed medical director of the Twin City Ordinance Plant Hospital in New Brighton in 1941.

He retired from National Guard service in 1944 and was appointed as state surgeon of the Minnesota Defense Force with rank of lieutenant colonel in 1946.

Survivors include his wife, Leah; four sisters, Mrs. Lucy Adams, Emma and Alma Aune, Duluth, Minn., and Mrs. Arthur Asplund, Milwaukee, Wisconsin.

HAROLD L. BOLENDER

Dr. Harold L. Bolender, fifty-five, prominent St. Paul physician, died January 6, 1958 at Midway Hospital in St. Paul where he had been hospitalized for nearly three weeks.

He had practiced medicine in St. Paul since his graduation from the University of Iowa in 1930. His special work included one year of internship at Ancker Hospital in St. Paul.

A native of Northwood, Iowa, he received his preliminary education in the public schools of that community.

Dr. Bolender was a member of the Ramsey County Medical Association, an active member of the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife, Hazel; two daughters, Jean and Sandra, both of St. Paul; his mother, Mrs. Lizzie Bolender of Northwood, Iowa; and two brothers.

GEORGE R. FUGINA

Dr. George R. Fugina, a physician and surgeon in Mankato and Madison Lake for nearly fifty years, died January 10, 1958. He was seventy-two years old.

He served on the staff of St. Joseph's Hospital since 1909 and was on the Immanuel Hospital staff since 1920.

Dr. Fugina had been practicing medicine in Mankato since 1924 when he moved from Madison Lake. He practiced in Madison Lake since 1909.

He was born in Fountain City, Wisconsin. He was graduated from the College of Physicians and Surgeons at the University of Illinois in 1908. He served his internship at St. Joseph's Hospital, Ashland, Wisconsin.

He was a life member of the Blue Earth County Medical Society, the Minnesota State Medical Association, the American Medical Association and the Catholic Order of Foresters.

His survivors include his wife, Maria; one daughter, Mrs. Lawrence A. Atwell, Arlington, Virginia; one sister, Miss Laura Fugina, Fountain City; and two great-grandchildren.

ANDREW W. HILGER

Dr. Andrew W. Hilger, seventy-eight, pioneer St. Paul ear, nose and throat specialist, died January 6, 1958.

A native of Bismarck, North Dakota, Dr. Hilger had practiced medicine in St. Paul for fifty years. He was a graduate of the University of Minnesota. Dr. Hilger was a member of the Ramsey County Medical Association and a life member of the Minnesota State Medical Association.

Surviving are his wife, Winnifred; two sons, Andrew W. Hilger, Jr., and Dr. John R. Hilger, both of St. Paul; a daughter, Mrs. Harold Thibault, Coronado, California; two brothers, Robert and John, and a sister, Mrs. Robert Prendergast, all of St. Paul; and two grandchildren.

ALVAH W. JONES

Dr. Alvah W. Jones, pioneer Red Wing physician, died January 4, 1958, at the age of ninety-four.

Dr. Jones was born in Fulton County, Ohio. He attended high school in Wauseon, Ohio, and came to Red Wing in 1882. He studied at the University of Minnesota for three years.

During the years of 1885 and 1886 Dr. Jones taught at Tower School and later became an instructor at the Hauge Seminary in Red Wing. In the fall of 1887 Dr. Jones enrolled at Rush Medical College and later that year accepted a position as special pension examiner for the United States government.

In 1888 he entered Georgetown University and was graduated as a doctor of medicine in 1891. He was valedictorian of his graduating class.

During his many years of practice in Red Wing, Dr. Jones served as city health officer for twenty-one years and for fifteen years was a member of the Red Wing board of education. He was also active in the Masonic Lodge, being a member of Red Wing Lodge, No. 8, A.F. and A.M.

Dr. Jones was a member of the Goodhue County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife and one son, Frederick W. Jones of San Leandro, California.

(Continued on Page A-50)

CLINICAL COLLOQUY

My patients complain that the pain tablets I prescribe are too slow-acting... they usually take about 30 to 40 minutes to work.

Why don't you try the new analgesic that gives faster, longer-lasting pain relief?

What is it... how fast does it act?

It's Percodan*—relieves pain in 5 to 15 *minutes*, with a single dose lasting 6 hours or longer.

How about side effects?

No problem. For example, the incidence of constipation with Percodan* is rare.

Sounds worth trying— what's the average adult dose?

One tablet every 6 hours. That's all.

Where can I get literature on Percodan?

Just ask your Endo detailman or write to:

Endo®

ENDO LABORATORIES

Richmond Hill 18, New York

*U. S. Pat. 2,628,185. PERCODAN contains salts of dihydrohydroxycodeinone and homatropine, plus APC. May be habit-forming. Available through all pharmacies

IN MEMORIAM

WILLIAM WINFRED KLIMA

Dr. William Winfred Klima, seventy-three, of Stewart, died December 11, 1957, in the Hutchinson Community Hospital where he had been a patient since July.

At the time of his retirement in August, 1956, he had been a practicing physician in the Stewart community for thirty-seven years.

A native of Silver Lake, he attended Hutchinson public schools. Prior to attending the University of Minnesota, Dr. Klima taught in rural schools in the Hitchinson area for several years. He was graduated from the University of Minnesota in 1917.

For one year after his graduation Dr. Klima interned at Ancker Hospital in St. Paul. He practiced for one year in his home community of Silver Lake. He opened his practice in Stewart in 1919.

Dr. Klima was active in the civic life of Stewart. He was a former member of the Stewart City Council. During World War II he was actively engaged in conducting community first aid courses.

He was a member of the McLeod County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife, Martha; two daughters, Mrs. Tom Koeppen, Hutchinson and Mrs. Howard Morton, Moline, Illinois; seven grandchildren; and one sister, Mrs. Frances Truhlar of St. Paul.

EUGENE T. LEDDY

Dr. Eugene Thomas Leddy, widely known specialist in x-ray treatment and a member of the staff of the Mayo Clinic for thirty-two years, died at St. Mary's Hospital in Rochester, Minnesota, on January 5, 1958. He had retired from active practice on October 1, 1957. Dr. Leddy was born in Taunton, Massachusetts. He received the degree of bachelor of arts in 1915 and that of doctor of medicine in 1919, both from Harvard University. He was an intern at Memorial Hospital in New York City for three months, and at Carney Hospital in Boston for 6 months. From 1920 to 1922 he was an assistant at the Memorial Hospital, and from 1922 to 1925 he was head of the roentgen-ray department of the Philadelphia General Hospital. For six months in 1925, he carried on the private practice of roentgenology in Providence, Rhode Island, where he was head of the x-ray department at the Rhode Island Hospital.

Dr. Leddy came to Rochester, Minnesota, on October 1, 1925, as a first assistant in therapeutic roentgenology. On April 1, 1929, he was appointed to the staff of the Mayo Clinic as a consultant in therapeutic roentgenology and as an instructor in radiology in the Mayo Foundation, Graduate School, University of Minnesota. He was advanced to assistant professor in 1933 and associate professor in 1937. He was certified as a specialist in radiology in 1934 by the American Board of Radiology, Inc. He was appointed head of the Section of Thera-

peutic Radiology at the Mayo Clinic on July 1, 1948, a post he held until April 1, 1953, when he became a senior consultant in therapeutic radiology.

Dr. Leddy, almost from the time of his graduation from medical school, devoted his career to the treatment of various conditions, especially malignancies, with x-rays and radium. He was regarded as a pioneer in the medical uses of radium.

Dr. Leddy was a fellow of the American College of Radiology and a member of the Zumbro Valley Medical Society, an associate member of the Minnesota State Medical Association, the American Medical Association, the American Roentgen Ray Society, the Radiological Society of North America, the American Radium Society, the Alumni Association of the Mayo Foundation and the Society of the Sigma Xi. He was an honorary member of the Old Blockley Medical Society of Philadelphia.

Dr. Leddy was married to Miss Anna C. Regan on June 8, 1926. Mrs. Leddy and two children survive him: John Eugene Leddy and Anna Marie Leddy.

PETER MILTON MATTILL

Dr. Peter Milton Mattill, seventy, assistant superintendent and associate medical director of Glen Lake sanatorium since 1938, died January 12, 1958 in Northwestern Hospital in Minneapolis.

Dr. Mattill, a resident at the sanatorium, had served on its staff since 1924.

Born in O'Daniel, Texas, he received his preliminary education at Albany, Missouri, and St. Joseph, Missouri, high schools. His bachelor of science degree was conferred at North Central College, Naperville, Illinois, and his master of science degree at the University of Chicago. He received his medical education at Rush Medical College in Chicago.

Dr. Mattill was a staff member of Children's Memorial Hospital, Chicago, and Rood Hospital, Hibbing-Chisholm, Minn., before joining the Glen Lake institution as a staff physician.

The American College of Physicians made him a fellow in 1929. He also belonged to American Board of Internal Medicine and American Trudeau Society. He was president of the Minnesota chapter in 1952-53.

Dr. Mattill was clinical assistant in medicine at University of Minnesota School of Medicine from 1926 to 1946 and instructor in the University's School of Nursing from 1946 to 1956.

His memberships include the Hennepin County Medical Society, Minnesota State Medical Association, American Medical Association, Minneapolis Society of Internal Medicine, Gethsemane Lutheran Church and Glen Lake School Board.

Surviving Dr. Mattill are his wife, Nora A.; four daughters, Mrs. John R. Holum, Minneapolis, Mrs. Herbert Strom, Red Lodge, Montana, Barbara and Phyllis Mattill, Oak Terrace; three brothers, Andrew, St. Joseph, Missouri, Charles, Missoula, Montana, and Henry, San Diego, California; and a sister, Emma Mattill, Upland, California.

General Interest

Dr. R. B. Skogerboe has announced that **Dr. Bruce Boynton** of Grafton, North Dakota, has joined the staff at the Karlstad Memorial Hospital.

* * *

Dr. Kermit Halvorsen joined the Mesaba Clinic in Chisholm the first of January.

* * *

Three Mayo Clinic doctors participated in programs at two sectional meetings of the American College of Surgeons in January. **Dr. James H. DeWeerd** collaborated in a panel discussion on automotive injuries at a meeting at Dallas, Texas. **Dr. Malcolm B. Dockerty** participated in a symposium on "Studies on Control of Spread of Cancer" at the same meeting. **Dr. John M. Waugh** of the Mayo Clinic participated in the three-day sectional meeting of the College at Jackson, Mississippi.

* * *

Dr. B. A. Gingold, Minneapolis, has been elected president of Asbury Methodist Hospital medical staff for 1958. **Dr. C. V. Kusz** is vice president and **Dr. S. R. Maxeiner, Jr.,** secretary-treasurer. Others named to the executive committee are **Dr. C. A. Neumeister,** chief of surgery; **Dr. D. R. Hastings,** chief of medicine; **Dr. M. E. Baker,** chief of obstetrics and gynecology, and **Dr. William H. Rock,** chief of general practice.

* * *

Dr. Frank H. Krusen, head of the Section of Physical Medicine and Rehabilitation at the Mayo Clinic, has been reappointed by Gov. Orville Freeman to the State Board of Health for a term ending January 1, 1961. This is Dr. Krusen's second term.

* * *

Dr. C. W. Truesdale has sold his interest in the Glencoe Clinic to **Dr. M. M. Howell** effective January 1. However, Dr. Truesdale will continue to hold office hours at the Glencoe Clinic until his office quarters are opened in Glenhaven which will probably be some time in February.

* * *

Dr. Richard M. Hewitt, head of the Mayo Clinic Section of Publications for fifteen years, has been appointed the third Alfred P. Sloan Visiting Professor at the Menninger Foundation at Topeka, Kansas.

* * *

Dr. Harold E. Miller was re-elected president of the medical staff of Northwestern Hospital. Others renamed to the staff are **Dr. Albert J. Schroeder,** secretary-treasurer; **Dr. Mark C. L. Hanson,** chief of medicine, and **Dr. Edgar Ingalls,** chief of obstetrics and gynecology. New officers are **Dr. Maynard C. Nelson,** vice president; **Dr. Harold Buchstein,** chief of surgery; **Dr. Northrop Beach,** chief of pediatrics, and **Dr. Frederick Hass,** chief of general practice.

* * *

The new offices of **Drs. M. A. and F. M. Burns** are located on Main Street in Milan. Formerly the Milan Hotel, the building was purchased some time ago by **Dr. F. M. Burns** and the first floor was remodeled for the doctors' offices.

* * *

Dr. John S. Lundy, senior consultant in anesthesiology in the Mayo Clinic, Rochester, and professor of anesthesiology in the Mayo Foundation, Graduate School, University of Minnesota, was the recipient of the second Award of Merit of the Horace Wells Club of Connecticut at the sixty-third annual meeting of the organization of the club in Hartford. The award was given to Dr. Lundy for his outstanding contributions to anesthesiology during his career.

* * *

Dr. J. S. Blumenthal, Clinical Associate Professor, Department of Medicine and Chief of Allergy Clinic, University of Minnesota Hospitals, has been appointed a member of the National Committee on Allergy of the American College of Chest Physicians.

* * *

Dr. Edward A. Pasek, Faribault, has recently been certified by the American Board of Ophthalmology.

* * *

Dr. Gordon Erskine, Grand Rapids physician, has announced his retirement after practicing in that community for almost twenty years. He is a former chief of staff at Itasca Memorial Hospital.

(Continued on Page A-54)

"ABOUT THIS JONES ACCOUNT, DOCTOR. WHAT ARE THE CHANCES FOR RECOVERY?"

Now available from Benson's...

the new Univis I.S./22 multifocal

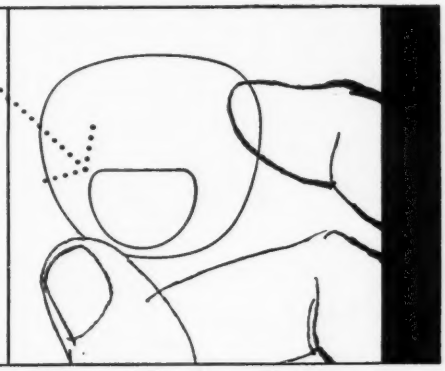
—the lens with the completely-identifiable segment

✳ Distinctive shape of segment gives positive identification ✳ Distinctive style protects you and your patients from imitation ✳ UNIVIS quality design and manufacture assures maximum patient satisfaction

CHECK THESE OUTSTANDING FEATURES OF THE I. S./22 LENS

- Straight top with rounded corners—for immediate identification
- Optical center located 4 mm. below the top of the segment for minimum displacement of image
- Barium crown segment assures freedom from annoying color aberration
- Unsurpassed front surface quality
- Corrected curves
- Accurate segment power

Of course . . .
famous UNIVIS quality throughout!



Dedicated to provide unexcelled Rx service to the ophthalmic professions for over 40 years

Benson OPTICAL COMPANY — *Executive Offices • Minneapolis 2, Minn.*

COMPLETE LABORATORIES CONVENIENTLY LOCATED IN UPPER MIDWEST CITIES

the AMERICAN
NATIONAL BANK OF SAINT PAUL
MEMBER FEDERAL DEPOSIT INSURANCE CORPORATION

ARTIFICIAL LIMBS
ORTHOPEDIC APPLIANCES
TRUSSES
SUPPORTERS
ELASTIC HOSIERY

FREJKA
Abduction Pillow Splint by the Original Maker

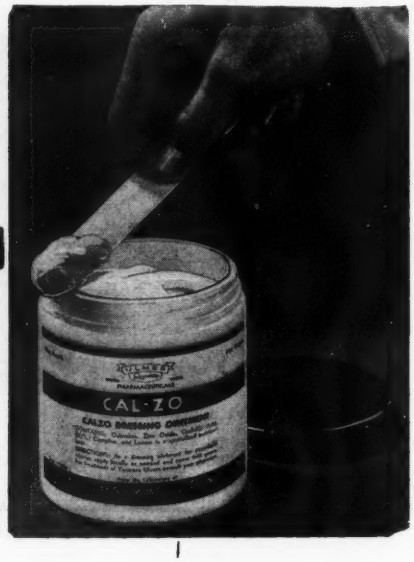
For displasia of the hip in the newborn and in early postnatal life (as described by Dr. V. L. Hart, Journal of Bone and Joint Surgery, Vol. 31-A, pp 357-372, April, 1949)

Prompt, painstaking service

BUCHSTEIN-MEDCALF CO.
223 So. 6th Street Minneapolis 2, Minn.

CAL-ZO

. . . still the best all-purpose dressing ointment



CAL-ZO (Ulmer), originally prepared as the most suitable dressing ointment for pressure-sponge ambulatory treatment of varicose ulcers, continues as the ointment of choice for dressings of every nature. CAL-ZO is ideal whenever calamine and zinc oxide are indicated.

CAL-ZO provides a semi-fluid cushion at body temperature thus protecting lesions under treatment. Contact between gauze threads and surfaces. CAL-ZO, because of its special base, will not soak into dressing at body temperatures. Easy to apply. CAL-ZO is the choice of the clinician when all-purpose dressing ointments are considered. Available in 1 oz. or 1¼ lb. jars. . . . MM-258b

The Ulmer Pharmacal Company

1400 Harmon Place Minneapolis 3, Minn.

(Continued from Page A-52)

Dr. Barnard Hall has been elected chief of staff at St. John's Hospital in St. Paul. **Dr. Paul Polski** was elected first vice chief of staff and **Dr. Paul Rick** was elected second vice chief. **Dr. M. G. Bernier** was named secretary. Elected to the executive committee were **Dr. A. E. Muller** and **Dr. C. R. Tift.** Dr. Hall announced appointment of **Dr. C. K. Cook** as chief of surgery, **Dr. M. E. Janssen,** chief of medicine; **Dr. Jack Strand,** chief of physical medicine and rehabilitation; **Dr. R. W. Emmons,** chief of obstetrics, and **Dr. Herbert Stolpestad,** chief of general practice.

* * *

Dr. John Pewters, president of the medical staff of Abbott Hospital, St. Paul, and the entire hospital executive board were re-elected for another year. **Dr. S. L. Arey** is vice president and chairman of the pediatrics division; **Dr. John Johnson,** secretary and chairman of medicine. Executive board members are **Dr. Tague C. Chisholm,** head of surgery; **Dr. Karl E. Sandt,** head of eye, ear, nose and throat division, and **Dr. Richard R. Fliehr,** in charge of obstetrics and gynecology.

* * *

"Medical Men of Meeker County" is the title of a book compiled by **Dr. H. E. Wilmot** of Litchfield.

* * *

Dr. Arthur Larson and his wife, **Dr. Dorette Larson,** Madison, have left the staff of the Fergus Falls State Hospital after serving there for eight months. **Dr. Arthur Larson** will spend three years at the University of Minnesota under a fellowship in pathology.

* * *

Dr. David E. Ellison, Minneapolis, has been named the president of the Twin City Rapid Transit Company.

* * *

Dr. R. B. Pierce, Renville physician, has been granted a patent for a device to hold fractured bones in place. The device was developed after long study and research and incorporates the use of stainless steel pins to anchor the bones at the fracture.

* * *

New officers of the West Central Minnesota Medical Society include **Dr. Irwin Oliver,** president, and **Dr. Carl Swendseen,** secretary-treasurer. Both doctors practice in Graceville.

* * *

The University of Minnesota has received more than three million dollars in medical research grants during the twelve months ending June 30, 1957.

The federal public health service granted $1,625,000 of the $3,060,000 total. Other grants brought the total federal government participation to $1,902,000.

Private foundations outside Minnesota contributed $606,482; and private sources in Minnesota contributed $469,388. The state legislature appropriated $82,500 for medical research.

* * *

Dr. Robert R. Kierland, consultant in the section of dermatology, Mayo Clinic, Rochester, has been elected secretary-treasurer of the American Academy of Dermatology and Syphilology.

Dr. **Richard Bardon** has been named president-elect of the St. Louis County Medical Society for 1959. Dr. **J. C. Feuling** was installed as president for 1958. He succeeds **Dr. Earl E. Barrett.** Other officers named were Drs. **W. H. Parker,** Chisholm, vice president; and **R. O. Bergan,** secretary-treasurer.

* * *

Dr. F. A. Meyerding will retire as executive secretary of the Minnesota Tuberculosis and Health Association April 1. His successor will be Mr. J. G. Neal, supervisor of health, physical safety and recreational education for the state of Minnesota.

* * *

Rochester's newly appointed civil defense director is **Dr. Winchell McK. Craig,** a retired Mayo Clinic neurosurgeon and retired rear admiral in the Naval Medical Corps Reserve.

* * *

A medical-surgical building will be named for **Dr. George E. Fahr,** head of medicine at Minneapolis General Hospital for twenty-four years, staff member since at the Anoka Hospital. Dr. Fahr was recently honored for his contribution in the development of the electrocardiogram.

* * *

The McLeod County Medical Society has elected **Dr. M. M. Howell,** president and **Dr. C. W. Truesdale,** secretary-treasurer. Both doctors are residents of Glencoe.

* * *

New president of the Winona County Medical Society is **Dr. Carl R. Heise.** He succeeds **Dr. Sidney O. Hughes.** Also elected were vice president, **Dr. L. J. Wilson,** succeeding **Dr. R. B. Tweedy;** secretary, **Dr. C. M. Johnson,** succeeding **Dr. Wilson,** and treasurer, **Dr. C. W. Rogers,** replacing **Dr. W. W. Haesly. Dr. L. I. Younger** was re-elected delegate to the Minnesota Medical Association and **Dr. H. W. Satterlee,** Lewiston, was re-elected.

* * *

Dr. Norbert O'Keefe, recently named chief of staff of the Olmsted County Community Hospital, will continue his practice in Spring Valley as well as being chief of staff of the hospital during 1958.

* * *

Mayo Clinic doctors who have recently retired include **Dr. Virgil S. Counseller,** head of the section of general surgery since 1928, and **Dr. Samuel F. Haines,** member of the clinic board of governors.

* * *

Dr. L. M. Eaton, head of the sections of neurology of the Mayo Clinic, has been elected president of the Association for Research in Nervous and Mental Diseases held recently in New York.

* * *

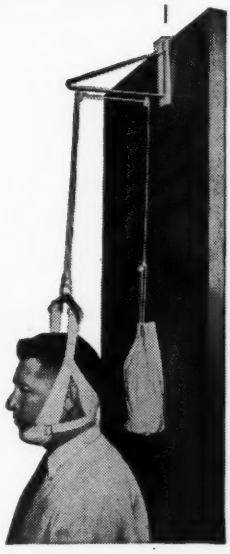
Dr. A. M. Lundholm, former St. Paul surgeon, is now associated with the Cambridge Clinic.

* * *

Dr. Richard A. DeWall, a Minnesota fellow in surgery at the University of Minnesota Hospitals, has been named one of the ten outstanding men of the year by the United States Junior Chamber of Commerce.

P&H offers this efficient, simple, easy to use

Head Halter Traction Kit



- Expertly designed and snug fitting
- Efficient, simple, easy to use
- Reasonably priced and complete as a kit

This Hand Halter may be used for many kinds of traction . . . cervical fractures, spasm of neck muscles, certain types of headaches, arm pains, types of spinal TB and many other uses. Each kit contains: bracket unit with rope, pulleys, sand bag and protective door pad; a head halter unit and a notched spreader bar unit. Available in adult or child size.

Ask your P&H Representative for complete information or write for brochure . . . MM-258a.

Physicians & Hospitals Supply Co.

1400 Harmon Place • Minneapolis 3, Minn.

Dr. DeWall distinguished himself for the discovery of a life-saving artificial heart-lung machine—called "bubble oxygenator." The device has been used in 357 open heart operations at University of Minnesota Hospital in the past two and one-half years.

* * *

Dr. C. A. Williams, Pipestone, has retired after devoting forty-five and one-half years to the medical profession. He has sold his practice to **Dr. R. J. Kotval,** who had been his partner since July 1, 1953.

* * *

Latest addition to the Olmsted Community Hospital staff in Rochester is **Dr. C. J. Corrigan,** a radiologist, formerly of Minneapolis and Saint Paul.

* * *

Dr. Ralph K. Ghormley of the Mayo Clinic staff recently participated in a discussion this month at the 18th annual Congress on Industrial Health in Milwaukee.

* * *

Dr. J. F. Haas, who previously was associated with **Dr. Glenn Nelson** in Fairfax, is now practicing with **Drs. S. T. Kucera** and **G. N. Rysgaard** in Northfield.

* * *

Dr. Harold S. Diehl has left University Hospitals at Minneapolis following his recovery from minor injuries suffered in an automobile accident the day before Christmas. Dr. Diehl, dean of the College of Medical Sciences at the University, suffered a dizzy spell at the wheel of his car and hit a bridge retaining wall. The car was badly damaged when it was wedged between the wall and a telephone pole. He suffered only minor injuries in the accident, but entered the hospital for a further checkup of his general condition. Recently, Dr. Diehl accepted the post as senior vice president for research and medical affairs and deputy executive vice president of the American Cancer Society. He has since taken over his new duties in New York.

AMA PRESIDENT-ELECT MAKES TWIN CITY APPEARANCES

"Medical Horizons" was the title of a convocation address presented by Dr. Gunnar Gundersen, president-elect of the American Medical Association at the fifty-sixth annual Farm and Home Week, held January 14-17 on the University of Minnesota St. Paul campus.

Minnesota's medical pioneers knew that good medical practice depends primarily on sound education, Dr. Gundersen told the convocation audience as he traced the various steps in the development of medical practice in Minnesota throughout the past century.

He also paid tribute to the Mayo Clinic at Rochester, Minnesota, saying the world-famed medical center grew out of the great character, human kindliness and profound sympathy of its founding brothers, Doctors Charles and William Mayo.

American society is discouraging bright young men from going into medicine, Dr. Gundersen told a press

TAKE A LOOK AT
NEW DIMETANE
THE UNEXCELLED
ANTIHISTAMINE

conference held in conjunction with his Twin City appearances. He emphasized unless something is done about it, America might find itself in the same situation as the Soviet Union, where eighty per cent of medical students today are women. The A.M.A. president-elect pointed out that immediately following World War II, there were five applicants for each opening in a United States medical school.

Today, he stated, there are less than two. However, he said, the situation after the war was unusual because there was a great backlog of young men whose college education had been delayed because of service. One of the things that make a medical center less attractive, is the growing encroachment of government, which is repugnant to independent, free-thinking Americans. One example he cited was government directed medical care of veterans. Medical care for service-connected disabilities properly is the concern of government, he said. But veterans should go to private practitioners for care of non-service-connected disabilities, he added.

He also cited the length of medical education—ten years—and its cost as factors which make young men hesitate about going into medicine. A medical education today costs $15,000, he said. Many young people feel they cannot afford such an investment, he continued, especially when they consider that the average productive life of a physician is twenty-five years.

Dr. Gundersen's recent Twin City appearance marks the first of several which he is expected to make in Minnesota during 1958. On June 14, he will present the address at the University of Minnesota Medical School's commencement exercises. During December, he will attend the American Medical Association clinical meetings which will be held in Minneapolis.

CORRECTION

In the Summary and Conclusion of the article entitled "A Practical Approach to the Suppression of Lactation," published in the November, 1957 number, pages 794-796, the dosage should read "*one* tablet three times a day for the first four days and one tablet daily for the ten days thereafter."

U.S. EXPECTS PHYSICIAN-DENTIST SUITS

(Continued from Page 137)

"It must have been an oversight," said the Court, "for it seems to us that any reasonable creature in being whose mind was not in a state of dover . . . would inevitably reach the conclusion that if plaintiff's services were to be continued after passage of the act of June 29, 1953, a commission would be required if the provisions of the law were complied with."

After June 29, 1953, the doctor-draft law required that inductees receive rank commensurate with experience and training. To date, the Army alone has uncovered eighteen potential plaintiffs.

Dimetane®
(PARAMINOPHENDAMINE MALEATE)
TABLETS (4 MG.), ELIXIR (2 MG. PER 5 CC.)
AND EXTENTABS® (12 MG.)
UNEXCELLED
POTENCY, UNSURPASSED THERAPEUTIC
INDEX AND RELATIVE SAFETY. MINIMUM
DROWSINESS AND OTHER SIDE EFFECTS.
A. H. ROBINS CO., INC., RICHMOND, VIRGINIA. ETHICAL PHARMACEUTICALS OF MERIT SINCE 1878.
Robins

MINNESOTA BLUE CROSS-BLUE SHIELD

The Professional Relations Staff of Minnesota Blue Shield traveled over 25,000 miles during 1957 to visit doctors' offices throughout Minnesota and to inform physicians on Blue Shield matters. More visits to physicians' offices during 1957 resulted in personal visits with doctors than in any previous year since the beginning of the Professional Relations Program in 1953.

While claim matters are most frequent inquiries of physicians involves the proper handling of medical service reports for patients who are subscribers with other Blue Shield Plans. Such services can be reported on Minnesota Blue Shield Medical Service Reports, which include the information required by other Blue Shield Plans. Reports should then be mailed directly to the Blue Shield Plans in which the patients are subscribers. If the address of any Plan is not available, or if any other information is needed, Minnesota Blue Shield will gladly provide assistance.

During 1957, many physicians inquired about the availability of the Blue Shield coverage for themselves and their families. In February of 1956 the Blue Shield Board of Directors made Blue Shield contracts available to doctors, because it was found that many doctors considered Blue Shield coverage a method of solving some of the embarrassments arising from the courtesy care of one physician by another. A doctor who wishes to obtain a Blue Shield contract can do so by writing to Blue Shield, 2610 University Avenue, St. Paul 14, Minnesota.

Another matter about which physicians ask is Blue Shield enrollment on a national level or wherever Blue Shield Plans are in operation. As of September 30, 1957, a total of 73 Blue Shield Plans were in operation in 43 states, the District of Columbia, Puerto Rico, Hawaii, and Canada. Total enrollment as of this date was 40,686,426.

Hospital admission figures recently compiled by Minnesota Blue Cross indicate that upper respiratory infections; pneumonia, flu and the common cold hit Minnesota citizens harder during the months of October and November, than at any other time in the past few years.

Nearly one-fourth of the 26,203 respiratory cases incurring Blue Cross claims through the first eleven months of 1956 occurred during the months of October and November. During October and November, a total of 6,029 Blue Cross members were hospitalized for upper respiratory infections.

The obvious upswing in respiratory infections proved to be costly. Blue Cross hospitalization expense for respiratory illnesses of $260,000 and $390,000 during the months of October and November, respectively, exceeded the average cost per month of approximately $200,000 experienced through the first nine months of 1957.

Over-all Blue Cross hospitalization expense through the first eleven months of 1957, totaled $25,969,419, providing 1,122,141.6 days of hospital care for 185,449 participant subscribers.

Accidental injuries incurred by Blue Cross subscribers still remain the principle cause of hospitalization with 31,434 cases representing seventeen per cent of total cases paid to date.

Pregnancies and conditions relating to pregnancy involved 29,681 subscribers and represented sixteen per cent of all cases paid during the first eleven months of the year.

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION

The Nineteenth Annual Meeting of the American Industrial Hygiene Association will be held in Atlantic City, New Jersey, April 21-26, 1958. More than one hundred technical papers will be presented on topics from the fields of radiation, air pollution, noise, toxicology, engineering control of hazards, and analytical chemistry. Professional hygienists and interested persons from industry, public health, labor and management groups, schools and universities, safety and technical organizations, medical institutions, and from the armed forces will convene to discuss current problems and recent advances in these fields.

Headquarters for the AIHA activities will be the Claridge Hotel. Registration will commence Sunday, April 20, at the Claridge Hotel and at Haddon Hall. Beginning Monday, April 26, registration will continue at the Convention Hall.

Advance registration may be obtained by mailing $3.00 to the Industrial Health Conference Registration, Room 1300, 28 E. Jackson Blvd., Chicago 4, Illinois.

Your one-stop direct source for the
FINEST IN X-RAY
apparatus... service... supplies

DIRECT FACTORY BRANCHES

DULUTH
928 East 2nd St. • RAndolph 4-8648

MINNEAPOLIS
808 Nicollet Ave. • FEderal 6-1643

RESIDENT REPRESENTATIVES

MANKATO
E. K. ADKISSON, 135 W. Gwyn St. • Phone 8-1568

MOORHEAD
R. N. MEYER, 1721 4th Ave., S. • Phone 3-4690

SIOUX FALLS
A. G. TROSTAD, 2501 Blauvelt Ave. • Phone 2-3066

Clinical experience[1,2,3] indicates that CELONTIN:

- provides effective control with minimal side effects in the treatment of petit mal and psychomotor epilepsy;
- frequently checks seizures in patients refractory to other medications;
- has not been observed to increase incidence or severity of grand mal attacks in patients with combined petit and grand mal seizures.

Optimal dosage of CELONTIN should be determined by individual needs of each patient. A suggested dosage schedule is one 0.3 Gm. Kapseal daily for the first week. If required, dosage may be increased thereafter at weekly intervals, by one Kapseal per day for three weeks, to maximum total daily dosage of four Kapseals (1.2 Gm.).

1. Zimmerman, F. T., and Burgemeister, B.: *Arch. Neurol. & Psychiat.* 72:720, 1954.
2. Zimmerman, F. T., and Burgemeister, B.: J.A.M.A. 157:1194, 1955.
3. Zimmerman, F. T.: *Arch. Neurol. & Psychiat.* 76:65, 1956.

the Parke-Davis family of anti-epileptics provides specificity and flexibility in treatment for convulsive disorders

for grand mal and psychomotor seizures

DILANTIN® Sodium (diphenylhydantoin sodium, Parke-Davis) is supplied in a variety of forms—including Kapseals® of 0.03 Gm. and of 0.1 Gm. in bottles of 100 and 1,000.

PHELANTIN® Kapseals (Dilantin 100 mg., phenobarbital 30 mg., desoxyephedrine hydrochloride 2.5 mg.), bottles of 100.

for the petit mal triad

CELONTIN® Kapseals (methsuximide, Parke-Davis), 0.3 Gm., bottles of 100.

MILONTIN® Kapseals (phensuximide, Parke-Davis), 0.5 Gm., bottles of 100 and 1,000. MILONTIN Suspension, 250 mg. per 4 cc., 16-ounce bottles.

DETROIT 32, MICHIGAN

24439